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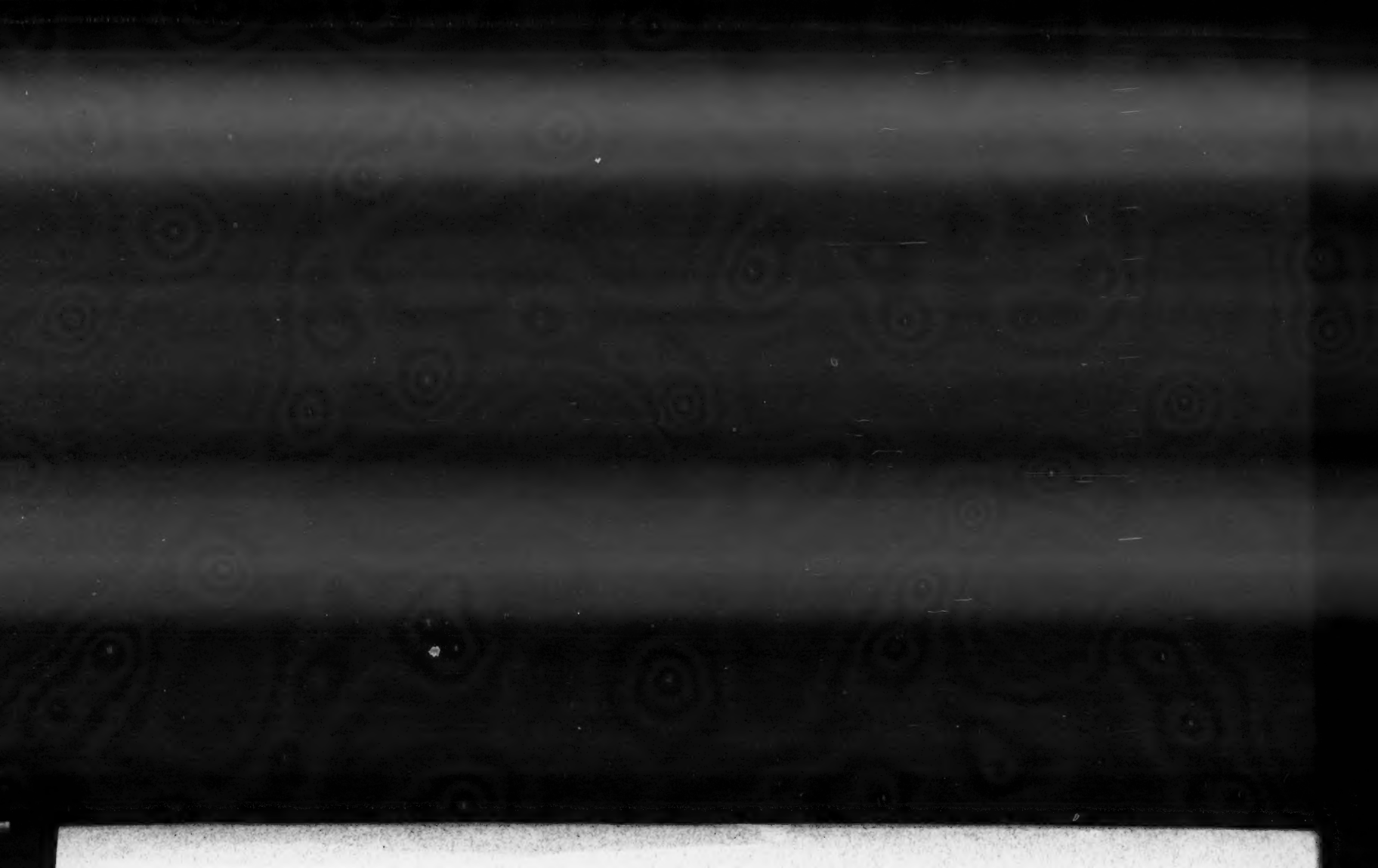

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LXXVIII.

FOCAL INFECTION AND ELECTIVE LOCALIZATION IN THE PATHOGENESIS OF DISEASES OF THE EYE.*

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In no field, perhaps, is there more convincing clinical evidence of the causal relationship of focal infection to diseases in remote parts of the body than in that of diseases of the eye. The importance of acute septic infections as a cause of metastatic disease of the eye and other organs has long been recognized. The idea, however, that mild, often symptomless, localized infections such as occur so commonly about teeth and in tonsils, sinuses or prostate may also predispose to diseases of the eye and other remote structures, while recognized by a few observers for a long time, gained little headway until the idea was placed on a sound basis about fourteen years ago by Billings. He first emphasized its importance on the

*Read as part of a symposium on Focal Infection before the American Academy of Ophthalmology and Otolaryngology, Detroit, Mich., September 13, 1927.

basis especially of a coordinated clinical and experimental study of cases. Since then great progress has been made. The idea is applicable to such a wide range of diseases and the mild localized infections occur commonly in so many structures that the quite general application of the principles involved has greatly modified the practice of medicine and surgery, including the various specialties so well represented in the Academy of Ophthalmology and Otolaryngology, and even the practice of dentistry.

In a large number of papers the importance of this factor as a cause of various diseases of the eye has been emphasized through the report of cases in which foci were demonstrable and relief attended removal of foci of infection. These and other corroborative facts leave no doubt of the correctness of the idea. Much still remains to be learned concerning the proportion of cases in which this factor is directly responsible and concerning the relative importance of the site of the focus or foci. However, from a study of the many reports already available, it appears that while localized infections in tonsils, sinuses and intestinal and genitourinary tracts are of great importance, especially since they may cause infection of teeth, those in and about the teeth appear especially prone to cause ocular manifestations.

While there is quite general agreement regarding the etiologic relationship of focal infection wherever found to diseases of the eye, there is still a difference of opinion regarding their nature, and whether they are due to localization of organisms or their toxins, especially in those that run a chronic course and that manifest periodic exacerbations and remissions. This is particularly true in those believed to be due to streptococci, because many clinicians still have the impression that all members of this group of organisms are highly virulent and that any disease in the eye due to localization of streptococci must needs be severe and associated with suppuration. This is erroneous, for it has been proved beyond doubt that in the streptococcal group more, perhaps, than in any other species of bacteria, there are individual strains of almost every degree of virulence. There are strains that affect, in diverse manner, a wide range of tissues or organs, while others have specific or elective affinity for particular structures without noticeably

affecting others. Clinical evidence in support of these experimental findings is not lacking, for in some of the diseases of the eye, such as "rheumatic" iritis, the ocular symptoms are only a part of similar manifestations in joints, muscles, heart and other structures, while in most of them the condition in the eye, as far as can be determined, is the only manifestation of disease. The pathogenesis of this group, especially the mild yet menacing affections, is still a puzzling problem. The difficulties of solution are many. Relief from symptoms following removal of foci of infection, while good evidence of causal relationship, still leaves open the question whether the lesions in the eye were the result of localization of bacteria or their toxins only. Material for cultural and microscopic study are not obtainable and, judging by experimental findings, even if the fluid in the anterior chamber were aspirated, it would likely be sterile when living organisms were present in the affected tissues. Systemic reactions are slight, cultures from the blood are negative, antibody production is minimal, and hence agglutination reactions with suspected organisms are of little or no diagnostic value. Antisyphilitic therapeutic tests and the tuberculin test cannot always be relied on to distinguish syphilitic and tuberculous from other forms of infection of the eye.

I wish now to consider the experimental results of the intravenous injection of bacteria from foci of infection, which throw more light, perhaps, than other methods of study on the mechanism and cause of nontuberculous and nonsyphilitic metastatic infections of the eye.

In connection with a long series of experiments in which animals were injected under uniform conditions with streptococci isolated from patients manifesting different systemic diseases, I have had the opportunity to study the lesions of the eye that sometimes developed in the injected animals. Unilateral panophthalmia was noted five times, twice following injection of the streptococcus isolated from the joints in cases of rheumatic fever, once with the streptococcus isolated from herpes zoster after passage through one animal, and once after the injection of streptococcus mucosus isolated from the pus in the eyeball in a case of metastatic ophthalmia following tonsillitis and otitis media. Hemorrhages with or without epi-

scleritis was noted in the region of the limbus in fourteen instances. Iritis or iridocyclitis developed in nine, four times after injection of certain laboratory strains, three times with streptococci from rheumatic fever and twice following injection of streptococci from small pus pockets in tonsils of normal persons. Conjunctivitis, associated with the streptococcus injected, occurred in ten instances and ulcer of the cornea in one. Hemorrhages in the posterior part of the eyeball were not often looked for but were found in two instances. The lesions of the eye were acute and consisted of edema and extravasation of erythrocytes associated with a variable degree of infiltration by leukocytes. Thrombosis of small vessels rarely occurred. The organism injected was demonstrated in the lesions in large or small numbers by cultural methods and microscopically in sections. This was true in some instances after the animal had seemingly recovered and cultures from the blood proved negative. The bacteria were found within blood vessels adjoining the lesions and free or within leukocytes in the areas of hemorrhage and infiltration. Suppurative iritis was found only once. The frequent occurrence of lesions in the ciliary body, iris and limbus in these animals parallels, roughly, what occurs in man. This might be attributed to the fact that here, as in the structure about joints and tendinous ends of muscles, there is a gradation from an abundant to a scanty blood supply, and hence a gradation in the supply of available oxygen, thus inviting localization and affording favorable conditions for growth of bacteria. This circumstance would also favor the production of lesions by bacterial or other toxins.

It must not be supposed that the occurrence of the lesions in the eye was wholly accidental, for they occurred more often with strains isolated from diseases in which affections of the eye are relatively common, such as in rheumatic fever and herpes zoster, than with strains isolated from diseases as in appendicitis, ulcer of stomach and cholecystitis, in which lesions of the eye are rare. The production of ophthalmia with the streptococcus isolated from the eye in cases of metastatic ophthalmia is also suggestive. These experimental results and many clinical facts indicate that localization may be dependent on inherent qualities of the various streptococci injected. Precise evidence in favor of this view was obtained some years

ago in connection with experiments on the transmutation of pneumococci and streptococci. In this work various strains of pneumococci and streptococci of low virulence were passed successively through animals by intravenous injection in order to raise their virulence, to note the changes in localizing power, if any, and to observe changes in cultural features and in morphologic characteristics. Thus, a stram which had been isolated eleven years previously as a pneumococcus and which had long since lost its virulence, was passed successively through seventeen animals without producing ocular lesions in a single instance; but, in the eighteenth passage, it produced suppurative conjunctivitis in the only rabbit injected. In the nineteenth passage it produced iritis (rabbit). In the twentieth passage it produced suppurative conjunctivitis associated with iritis and marked hemorrhages and infiltration at the limbus in a dog, and marked conjunctival and scleral hemorrhages, especially at the limbus, in a rabbit. Subsequent passages failed to produce ocular lesions. Similar results were obtained from injection of two strains of pneumococci in successive animals. At the time, these grew as streptococci, and had lost their virulence by long cultivation on artificial media; each produced iritis in the tenth and fourteenth passage through an animal, while all the other injections failed to produce lesions of the eye. The injection of a strain which originally had been isolated as a hemolytic streptococcus from "milk slime," but which had lost its hemolytic property and had acquired the properties of typical streptococcus viridans by growth in symbiosis with bacillus subtilis on a blood agar plate, produced no lesions in the first seven injections, but in the eighth passage through an animal produced hemorrhages about the limbus and several in the retina in two rabbits injected; in all subsequent passages no ocular lesions developed.

The results of these animal experiments, in which marked changes in localizing power in different structures were noted, furnished the experimental basis for the theory of elective localization and led to an extended study, under a constant technic, of the localizing power of bacteria isolated from foci of infection and systemic lesions in diseases affecting widely different structures and of widely different character. Striking evidence of specific or elective localizing power corresponding

in situation to the organs or structures involved in the patient was obtained in many of these, including diseases of the eye. The importance of strict attention to technical details, such as the proper selection of cases, use of partial tension cultures and the injection of freshly isolated cultures, became apparent and has since been repeatedly emphasized by myself and by Haden.

In 1916, Irons, Brown and Nadler reported the successful production of iridocyclitis in rabbits by the intravenous injection of streptococci isolated in cases of acute iridocyclitis. They were able to show that the power of the streptococcus to invade special tissues may change within a short time during residence in the host, during passage through an animal, and in cultures.

Numerous attempts have been made, with only partial success, to find a method that might be relied on to preserve elective localizing power and viability for long periods. Preservation of latent life under reduced oxygen tension in tall tubes of meat mash infusions or in shake cultures containing relatively few colonies in glucose brain agar or ascites glucose agar, in dense suspensions in glycerin (two parts), and saturated sodium chlorid solution (one part), have often served to preserve viability and elective localizing and other specific properties for a long time. In exceptional instances this was true for as long as eight years, when the corresponding strains, grown aërobically, had lost elective localizing power during several daily transfers. Subcultures rapidly made (from four to eight each 24 hours), in previously warmed mediums which afford a gradient of oxygen tension, such as glucose brain broth, often serve to maintain specific localizing power and agglutinating properties for many generations. Elective localizing power of streptococci was preserved often for many weeks or months within the pulp chamber or periapical tissues of artificially devitalized and infected teeth in dogs.

The use of proper cultural methods is not the only requirement for the successful application of the principles involved in the study of elective localization, especially as pertains to localized inflammatory processes in different diseases. Here dif-

ficult procedures and judgment of elusive factors are often necessary. We have learned that a mere correct diagnosis may not be sufficient. The condition at the time of study should also be considered. A higher incidence of localizations and more marked lesions in animals are obtained if cultures are secured at the time of acute systemic manifestations or during exacerbations of symptoms in chronic conditions than during quiescent intervals. Great care is exercised in the collection of material for study, both as regards the clinical observations and the prevention of contamination by extraneous organisms. Pus from the depths of tonsils is expressed and cultures made from this instead of swabbings from the surface or from single crypts. The urethra is irrigated with sterile solution of sodium chlorid before the prostate is massaged to obtain material for cultures. Material from the nasopharynx is obtained by swabbings high behind the palate, with cotton swabs on aluminum wire bent to a suitable angle, without touching the tongue. Pus from the depths of pyorrhea pockets or discharging sinuses is aspirated into fine pipettes after the surface has been cleansed with alcohol or other antiseptic. Pulpless teeth are extracted under as nearly sterile condition as possible, and, in special instances, the apical end is resected for culture before the tooth is extracted. Culture controls are made of the mucous membrane before and after sterilization, and of the overlying bone after the mucoperiosteal flap is resected.

An opportunity to determine whether streptococci from foci of infection in diseases of the eye possess elective localizing power, and hence produce lesions in the eye, was afforded in April, 1919, in a case of recurring iritis, which was reported by Benedict. The clinical findings and results of the cultures and animal experiments are representative of others studied since by myself, by Benedict and by my associates. The patient, a young woman, was seen during the early stages of an attack of iritis similar, in every respect, to others which had occurred previously. The general examination was negative. Radiographs of the teeth revealed four impacted, unerupted molars and fracture of the roots of both upper central incisors, sustained by a fall eleven years before. The impacted and fractured teeth were extracted and the sockets of the incisors curetted. Recovery from the iritis was rapid. Smears from

the foul smelling pus found in the enlarged pulp chamber of one incisor showed gram positive diploccoci and gram negative fusiform bacilli. Aërobic cultures remained sterile, while partial tension cultures in tall tubes of glucose blood broth yielded an anaërobic streptococcus which produced iritis in a series of rabbits. Cultures from the turbid fluid from the anterior chamber remained sterile, while those from emulsions of the rabbit's iris yielded the organism injected; this again produced iritis on intravenous inoculation. In several of the rabbits hemorrhages had also occurred into the dental nerves and pulps and into muscles. The streptococcus was demonstrated microscopically in the lesions in the eye and proved absent in the unaffected tissues. Thus, by the use of partial tension cultures, it was possible to isolate a streptococcus with which the disease of the patient was reproduced; if aërobic cultures only were made, as is still so often the case, the teeth would have been pronounced sterile.

At about the same time, a similar partial tension streptococcus which produced lesions of the eye and of the pulps and nerves of teeth, was isolated from an inflamed dental pulp in the tooth of a man who had suffered from recurring attacks of rheumatic pains in the back, arms and shoulders and through the head.

Lewis during the same year produced iritis in a rabbit by injecting a culture of streptococcus viridans isolated from a periapical infection in a patient suffering from acute iritis.

Meisser and I have since found a high incidence of elective localization in the eyes of rabbits injected intravenously with streptococci from dental and tonsillar infections in patients suffering from acute iritis, and which was only rarely obtained with streptococci from chronic cases of iritis or other chronic affections of the eye.

In 1921, I reported the results of a study of two cases in which the eyesight had been well nigh lost from recurrent intraocular hemorrhages and in which focal infection had not previously been suspected as a causative factor. The method of intravenous injection of cultures into animals directly from foci of infection yielded a striking illustration of the mechan-

ism of the occurrence of this most deplorable of ocular conditions.

The cultures for injection in one were obtained from the vital pulp of a badly decayed tooth; in the other, from a suppurating sinus surrounding a third molar. Both yielded positive results. Of the nine rabbits injected, lesions of the eye developed in six. In one, panophthalmia with hemorrhages in the retina resulted. In the others, swollen, whitish areas and hemorrhages in the retina were demonstrated with the ophthalmoscope or by microscopic examination of sections. Streptococci were demonstrated in the lesions. Similar experiments made in other cases at the same time produced no lesions of the eyes. Clinical evidence of a direct connection between focus and ocular condition (in this instance probably due to absorption of toxin) was present in the case in which the suppurating sinus was found in the jaw. The degree of vision fluctuated, in a remarkable manner, with the stoppage or drainage of the pus exuding from the tooth. With lack of drainage, even for a day, the vision diminished on repeated occasions and increased when drainage was again established.

Benedict, in 1921, reported the clinical and experimental results in a series of cases indicating that the ocular lesions attributable to focal infections, especially various forms of iritis, are associated with localization and growth of organisms in the lesions and are not due to toxins.

Haden, in 1923, in a long series of experiments in cases of acute iritis, episcleritis, uveitis and hyalitis, has had similar results. He reported finding lesions of the eye in 68 per cent of sixty-six rabbits injected with freshly isolated cultures from teeth of patients suffering from metastatic infection of the eye. Of the control group in which parallel injections were made of cultures from infected teeth in persons that were well or were suffering from some other condition, lesions of the eye developed in only 14.8 per cent of 169 rabbits injected. He also emphasized the importance of the use of proper technic in order to avoid misleading results, and, among other conclusions, stated that "teeth which are negative in the radiograph may bear as important an etiologic relation to disease as those which show evident infection." The ocular lesions

induced in the animals resembled, in many respects, those noted in patients as regards clinical appearance, structures involved and microscopic changes, and were due in all instances to localization of the organism.

Nickel has reported the results of an extensive study (under a constant technic) of the localizing power of bacteria from foci of infection in tonsils, teeth, prostate and cervix, in patients suffering from various diseases, including diseases of the eye. The factor of specificity or elective localizing power was brought out in a striking manner in each of six groups of cases studied. Moreover, he found what I had repeatedly observed, namely, that the best results are obtained in cases of acute disease or during periods of exacerbation in cases of chronic disease. His results from a routine study in which all cases were included and in cases selected according to the type of focus at hand and acuteness of symptoms, as well as in control cases, are strikingly brought out in the tabulation. The high incidence (75 per cent) of animals that showed lesions of the eye in the selected cases is especially noteworthy; such lesions were found in only 34 per cent in the routine cases and in 1 per cent of the control group. In this connection the fact should be mentioned that he has found that lesions of the eye, especially congestion of vessels of the iris and circumcorneal region, have no doubt been missed by others; such congestion sometimes occurs and disappears within from six to twleve hours after intravenous injection and may not progress to exudation and hemorrhage. Moreover, in a series of as yet unpublished experiments, he has been able to show that cultures of streptococci having elective affinity for the eye produce a filterable toxin that affects specifically the eyes of rabbits when injected intravenously. Control experiments with filtrates of streptococci having no affinity for the eye have little or no effect. I have obtained similar results with filtrates of cultures of a bacillus and diplococcus isolated from the eyes of horses affected with periodic ophthalmia.

Benedict, Von Lackum and Nickel have shown the importance of streptococcal infections in the prostate and cervix in relation to diseases of the eye. They have reported clinical results in a series of cases and the results of the injection of

animals with freshly isolated cultures. Of twenty-four cases studied experimentally, fourteen yielded positive results in animals. Lesions of the eye developed in 29.5 per cent of sixty-one rabbits injected. Excluding cases in which the condition of the eye was chronic, the focus questionable and the symptoms quiescent, lesions in the eye were produced in 67 per cent of the animals injected. This was in sharp contrast to the incidence of 7 per cent of 181 animals injected with seventy-nine strains obtained from the teeth, tonsils, prostate and cervix of patients whose conditions were not definitely diagnosed and who presented no ocular symptoms. The authors found that the causative organism isolated from these foci was a slightly hemolytic or green producing streptococcus requiring partial tension methods for its isolation, and that local treatment and autogenous vaccines made from the strains obtained from experimental lesions of the eye in rabbits often greatly benefited the patient.

The underlying physicochemical conditions that determine elective localization of living bacteria are undoubtedly similar to, yet as obscure as, those that determine the specific pharmacologic action of drugs and chemicals. However, the fact that certain streptococci possessing elective localizing power, especially those from the lesions in ulcer of the stomach, myositis, epidemic hiccup and encephalitis, and now from iritis and other ocular infections, have been shown to produce poisons or toxins free in broth cultures and within the bacterial cell which have the power to injure electively the tissues or organs in which the respective living bacteria localize and produce lesions, throws a clearer light on how the bacteria overcome the inherent resistance of tissues against invasion.

SUMMARY AND CONCLUSIONS.

The more important clinical and experimental studies on the pathogenesis of nonsyphilitic and nontuberculous intrinsic infections of the eye and the requirements for the successful application of the methods of study are reported. The method of intravenous injection of primary (often mixed) or freshly isolated pure cultures of material from foci of infection has led to a better understanding of how these seemingly harmless localized areas of infection, often small and in obscure places,

cause ocular manifestations; and by this method the causative organisms usually responsible have frequently been isolated.

In the light of the newer knowledge, foci of infection, wherever found, should be looked on as a place where favorable conditions are afforded for entrance into the blood or lymph stream of bacteria and their toxic products and where they may acquire or maintain peculiar or relatively high invasive power. They make for a forced relationship between parasite and host.

The good effects commonly noted following removal of foci of infection support the experimental findings, justify a thorough consideration of their existence and call for removal or cure, as far as possible, of focal infection in every obscure clinical case. The successful application of the methods of study, while simple, require unusual facility of cooperation between bacteriologist and clinician. The experimental results indicate clearly that those lesions in the eye which are associated with exudation, even though slight, are usually due to actual localization of microorganisms, while the milder manifestations may sometimes be due to absorption of toxins which are formed in the focus or elsewhere and reach the eye in the blood stream.

Localization of the bacteria in the eye may sometimes be accidental and a part of other disease manifestations. However, the animal experiments, now amply corroborated, indicate clearly that localization and growth in most instances are due to peculiar, acquired or inherent, properties within the bacteria themselves and to their power to localize electively, and that this is, in part, due to the production of a toxin or poison which affects specifically the tissues in which localization and growth occur.

Among the microorganisms isolated which manifested the greatest elective localizing power and with which the common forms of diseases of the eye have been reproduced, is a streptococcus which usually produces greenish or slightly hemolytic colonies on blood agar, which requires a reduced oxygen tension for its isolation and from which autogenous therapeutic vaccines, of great value in many cases, have been prepared.

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TABULATION

Comparison of Results in Animals Injected With Cultures From Routine and Selected Cases (Nickel).

	Selected cases	Animals injected	Positive animals	Percentage of animals showing specific localization: Selected cases	Control cases	Routine cases
Arthritis	82	220	173	77	7	51
Myocarditis	9	29	18	62	3	39
Myositis	10	23	16	69	4	38
Lesions of the eye	11	24	18	75	1	34
Lesions of the skin	4	8	4	50	1	36
Peptic ulcer	39	116	80	68	3	40

LXXIX.

THE ETIOLOGY OF FOCAL INFECTION: MEDICAL ASPECTS.*

By Russell L. Haden, M. D.,

Kansas City, Kans.

The systemic expression of a focal infection is the result of the interaction of focus and patient. Here, as in most other infections, the activity of the infecting bacteria and the resisting powers of the infected individual are of prime importance. A discussion of the medical aspects of etiology of focal infection must necessarily be limited largely to a consideration of these two factors.

Before these factors are discussed in greater detail, one point demands the greatest emphasis. This is the need for a general medical survey of every patient presenting evidence suggestive of systemic disease of focal origin. Such a survey necessarily includes a carefully taken history, a competent physical examination, and such laboratory procedures as are indicated. In this way, the possibility of systemic disease being related to a focus of infection can usually be determined and the relative importance of the different factors be evaluated. The internist is constantly seeing patients who have suffered at the hands of specialists for the lack of such a survey. Often haphazard therapeutic procedures are undertaken without a clear perception of the problem in mind. Two patients seen very recently illustrate the truth of these statements. One patient had had a number of teeth removed for pain in the back. No relief was obtained, since the pain was due to a stone in the kidney. Another patient had all the teeth extracted for weakness. Again no relief followed, since the symptoms were due to an unrecognized pernicious anemia which probably has little relation to focal infection. The ob-

*Read as part of a Symposium on Focal Infection before the annual meeting of the American Academy of Ophthalmology and Otolaryngology, Detroit, Mich., Sept. 13, 1927.

serving internist in such a survey also keeps in mind the ever present possibility that symptoms commonly ascribed to focal infection may be due to very different causes. A toxemia of intestinal origin and other metabolic derangements may produce disease clinically identical with that characteristic of focal infection. A differentiation is often difficult to make.

Every careful student of focal infection is impressed with the fact that no subject in medicine must be handled with more mature clinical judgment when he sees the frequent examples of the harmful results of the reckless removal of foci, especially the teeth, the frequent ascribing of conditions to focal infection when due to entirely different causes, and the careless promises for the relief of systemic disease by the removal of foci. The more thoroughly one studies the subject, the more cautious one becomes. This does not alter the conclusion that focal infection is of the greatest importance in the causation of systemic disease.

Having determined that a patient's symptoms are probably due to a focal infection, the primary factors of focus and individual must be considered and evaluated. The first problem is to find the focus. Often this is apparent. In other cases it is very difficult to find. The focus may be located in many different places in the body. Theoretically a focus may exist at any point in the body in which a localized area of infection may persist under pressure. Cases are reported in which a chronic localized infection around a toe nail or in some other obscure site has resulted in serious metastatic disease. From a practical standpoint, only the teeth, the tonsils, the sinuses, the gastrointestinal tract, the prostate and seminal vesicles and the female generative organs need be considered. This seems to be a large field, which may, however, be still further delimited by the statement that the last three named are relatively infrequent sites of foci of clinical importance in systemic disease.

In every individual, however, suffering from systemic focal disease, all possibilities must be evaluated. The probability of each possible site will vary with the individual studied. Certain general principles which may aid in determining the focus. In young individuals metastatic disease arises very uncommonly from the gastrointestinal and genitourinary tracts. At this

age period the infection is most apt to arise from lymphoid tissue, hence the tonsils are most frequently at fault. In older individuals the other possibilities must be considered, although here also the prostate, seminal vesicles and female generative organs are of minor importance, considering the field as a whole.

The tonsils may be a focus at any age and should never be eliminated as a possible focus on the basis of negative clinical findings. Infection in sealed off crypts occurs very commonly. In the presence of manifest systemic disease of focal origin, a tonsillectomy is always indicated if the tonsils are not already removed, provided there are no contraindications for operation. The same may be said for tonsillar remnants which are frequently encountered after the tonsils have been removed. Infection in sinuses may occur at any age interval and give rise to metastatic disease.

Considering all cases of metastatic disease of focal origin, the teeth certainly hold first place as a source of infection. Infected teeth are almost universal, due to the widespread occurrence of dental caries and the practice in the past of killing pulps. Dental infection is especially responsible for systemic disease of focal origin occurring after middle age and for the diseases resulting from continuous damage over a long period of time, such as myocarditis. In a large group of individuals of all ages in Kansas City, I found 92 per cent had one or more pulpless teeth. An average of 1.4 abscessed teeth per person was found.

The development of systemic disease from a focus seems dependent in large measure on the conditions under which the bacteria live in the focus. This fact must determine in large measure the acquirement of pathogenicity, the condition in bone being especially favorable for the development of pathogenic properties. It is probable that in most instances at the time bacteria first enter a focus they have little pathogenicity. Living in tissues deficient in oxygen aids materially the development of specific toxins. Rosenow showed many years ago that the toxins of the pneumococcus, to which the streptococci of focal infection are closely related, develop more readily in the absence of oxygen. In experimental work, specific localiz-

ing properties can be preserved only under relatively anerobic conditions.

Physician and dentist agree that teeth showing radiographic evidence of infection should be removed. There has been much difference of opinion concerning the disposition of pulpless teeth which do not show radiographic findings characteristic of infection. Bacteriologic studies have shown that there is little difference in the frequency of infection in the two groups. If the removal of all possible foci is indicated, no pulpless teeth should be allowed to remain in the mouth.

In trying in a given case to determine, in the presence of several possible foci, which one is probably the most important factor, some information may be gained from knowing the type of systemic disease present. Thus, acute rheumatic fever, chorea, and rheumatic endocarditis and myocarditis arise probably entirely in lymph adenoid tissue. Other types of endocarditis or myocarditis may come from any focus. Acute glomerulonephritis is most apt to come from the tonsils. Other types of kidney disease, such as pyelonephritis, may come from other foci. Chronic infectious arthritis, on the other hand, comes by far more frequently from dental infection than from other sources.

A very important factor in focal infection is the multiplicity of foci. One constantly sees patients who present a number of possible foci. It may be proven by animal experimentation that even in the presence of multiple foci only one may be causing the systemic trouble. This can be determined only experimentally, however. Often it is difficult to determine the order in which foci should be removed. This will depend in part on the systemic manifestations. It is unwise to proceed too rapidly. In deciding precedence of removal between tonsils and infected teeth, usually the dental infection should be removed first. The only safe rule is to remove all possible foci if practicable.

The other aspect of the question of etiologic factors is the reaction of the host to the invading organisms. An individual may harbor one or more foci of infection without ever developing demonstrable systemic disease from the focus or foci. Another person with only a single focus, seemingly much less important, may develop serious disease. This great difference

in susceptibility is impossible to foresee and difficult to evaluate. It is probably constantly varying in every individual. Among the factors influencing it are the presence of chronic debilitating disease, such as nephritis, and the occurrence of an acute infection, such as measles or influenza. Age is also a most important factor. Especially after middle age is the resistance to infection on the wane. Even with a fall in resistance, however, a patient cannot develop systemic disease of focal origin unless a focus be present. I often use this simile in explaining to patients the importance of removal of foci: Suppose two individuals are exposed simultaneously to diphtheria. One is susceptible to the disease and contracts the infection, the other is immune and does not develop diphtheria. Here the determining factor is the degree of resistance; yet the susceptible individual would not develop the disease if he had not gotten in his throat the diphtheria bacillus. In the same way, a person may develop arthritis due to low resistance, yet the important factor is a focus, without which the disease of focal origin could not develop. Often little can be done to influence the resistance. Much can be done in eliminating foci.

The correct recognition of the factor of resistance in focal infection materially influences the prognosis. Given two patients with focal systemic disease of equal intensity, a better clinical result is to be expected from the removal of a virulent infection in an individual with good resistance than from the removal of a mild infection in an individual with low resistance. Two patients suffering from arthritis, seen at the same time recently, illustrate well this point. One was a young man on crutches with a severe subacute arthritis following a marked alveolar infection. The other was an elderly lady, who had developed an arthritis following the shock of a fall in which an arm was broken. She too had every indication that the focus of the joint trouble was in dental infection. In the first patient, the removal of the infected teeth enabled the patient to lay aside his crutches the next day. An excellent result was to be expected. In the second patient, the removal of the teeth had little effect on the course of the arthritis. Little was to be expected.

Foci of infection should not be disturbed unless there is something definite to be gained from the operation, either in preventing further disease or in aiding in the cure of sys-

temic disease of focal origin. The patient's physical condition may contraindicate any operative procedure. The systemic disease may be all out of proportion to the local lesion. In other patients, the structural changes may be beyond repair and of so serious a nature as to make the local lesion a minimal part of the clinical picture. Here it is often unwise to stir up foci even when causally related to systemic disease. Caution should be exercised in recommending the removal of foci in elderly individuals unless manifest advantage is to be gained thereby.

One more point is, it seems to me, of paramount importance. The recognition of the rôle of chronic foci of infection in systemic disease has been a gradual one. It has been brought about by the clinical study of the end results of such localized infections and by animal experimentation. We are now continually seeing patients suffering from systemic diseases of focal origin and attempting to cure them by removal of the focus or foci. So often the curative attempts are most disappointing. The symptoms of the systemic disease often represent the effects of the bacterial poison over a period of years and are dependent upon organic changes in tissue which cannot be undone. The realization of these facts serves only to emphasize the early removal of foci of infection. We should rather try to prevent the development of systemic disease by removing foci rather than attempt to cure systemic disease by this procedure. Especially with the increasing interest in periodic health examinations, more opportunities will be given for the early detection of foci and for giving the proper advice concerning them.

I may summarize what I have attempted to discuss in a few words.

The first requisite in the management of focal infection is a sane general survey of the patient, keeping in mind the possibility that the symptoms often ascribed to focal infection may be due to entirely different causes.

In the vast majority of cases, the foci are found in the teeth, tonsils and sinuses.

In determining the disposition of foci, factors influencing the resistance of the individual must be evaluated and the structural damage already done must be considered.

Very often the systemic disease is dependent upon continuous damage over a long period of time. Eradicating the foci may avail little.

Where the removal of foci is indicated, all possible foci should be removed.

The experimental and clinical study of focal infection emphasizes the important relation of foci to systemic disease and the need for the early recognition and removal of foci before systemic disease has resulted.

The systemic disease should be cured before it begins.

UNIVERSITY OF KANSAS SCHOOL OF MEDICINE.

LXXX.

OTOLARYNGOLOGIC PHASE OF FOCAL INFECTIONS.*†

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In a symposium of this character it is essential that each participant confine his remarks as closely as possible to the portion of the subject assigned to him. I, therefore, will endeavor to limit myself to the ear, nose and throat phase of this fascinating subject.

My experienec has been chiefly clinical, and it is from that point of view that I present these observations.

The numerous articles on this subject attest the widespread interest of the profession in the relation of focal infections to general diseases. The work of Billings and Rosenow stands out prominently in placing the subject upon a scientific basis, because of their careful clinical observations, confirmed by closely checked bacteriologic studies and extensive animal experimentation.

So many of these foci of infection are in the ear, nose and throat that this subject is of the greatest importance to the otolaryngologist.

Keiper[1] lists nearly seventy diseases in which the tonsils have been considered the offending focus, and this list by no means includes all of the diseases which other writers have attributed to the same cause.

Statements such as this may well make us pause and ask whether we are not being carried away by our enthusiasm beyond the limits of prudence.

Speaking of the clinical phase of focal infections, D. J. McCarthy,[2] of the University of Pennsylvania School of Medicine, says:

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*Read as part of a symposium on Focal Infection before the American Academy of Ophthalmology and Otolarngology, September 13, 1927, at Detroit, Mich.

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"In studying the influence of focal infections we should fully realize that we are dealing largely with theoretical considerations and not with a definite demonstrable fact, or series of facts, subject to a testing process such as the pathologist or bacteriologist gives out in a laboratory experiment. Any data on this subject necessarily must be largely a matter of impression on the part of the investigator, tinged somewhat by his state of mind toward the problem under consideration. In the vast majority of cases, the focal infectious process acts on an already existing condition of undernutrition, anemia, endocrin imbalance, etc.—more especially in individuals with arterial hypotension than those with normal blood pressure or arterial hypertension."

The work of Dr. Amy Daniels, at the University of Iowa, who is able to produce, at will, infection in the paranasal sinuses of rats, by a diet deficient in fat soluble viatmin A, while the control animals remain unaffected, calls attention to the importance of nutrition in this question of focal infections.

These observations emphasize the fact that this problem involves not merely the discovery and removal of a focus of infection, but the study of a diseased patient, requiring the closest cooperation between the otolaryngologist, the internist, the dentist and the bacteriologist, and the closer this cooperation, the better will be the result for our patients, and the more exact will be our knowledge of the subject now under discussion.

These general factors also probably account for the fact that many of us harbor foci of infection without developing any of the diseases commonly attributed to this cause, and that frequently such diseases undergo resolution without the removal of any offending focus, while in other cases the removal of all discoverable foci fails to result in clinical improvement.

METHODS OF INFECTION.

Apart from direct extension to surrounding tissues, infections in the upper respiratory tract may invade other structures by the direct inhalation or swallowing of the infective organism, by the entrance of toxins or the organism itself into the blood stream or into the lymphatic system.

TONSILS.

That infected tonsils may be the cause of general diseases is so universally accepted that their removal, either as a curative or prophylactic measure, requires no defense. The question of what constitutes a diseased tonsil is one that the laryngologist must decide for himself in each individual case. The author's opinion is that if there is a history of previous infection of the tonsils, or clinical evidence, such as induration of the tonsil tissue, redness of the anterior pillars or expressible pus, the patient will be benefited by their removal, especially if consultation with the internist discloses any of the general diseases in which a focus of infection may be considered a factor.

ADENOIDS.

That the adenoids may harbor infectious pus, which is not discoverable by the use of the ordinary postnasal mirror, is easily demonstrated by the use of Kelley's direct vision adenotome. This instrument frequently enables one to observe pus being squeezed out of apparently simple hypertrophied adenoids during their removal.

SINUSES.

The discovery of foci of infection in the paranasal sinuses is often quite simple. A mere inspection of the nasal cavity may disclose the presence of pus, and the source from which it comes is determined with comparative ease.

In other cases, however, the most painstaking care in repeated examinations, with the use of every available aid, such as transillumination, X-ray plates taken from different angles, the use of lipiodol by displacement instillation, as described by Proetz, and even the exploration of the sinuses themselves may be necessary before the presence of an infective focus can be decided. In children, sinus infection is quite apt to be overlooked, and the use of the X-ray and irrigation of the antra will frequently show an infection which routine inspection would not disclose.

The discovery of an infection in the paranasal sinuses does not necessarily require radical surgery, as many of these cases will clear up under conservative treatment, and this should

be tried if the general condition of the patient will permit. In children, especially, the removal of tonsils and adenoids and the use of ephedrin in the nose is frequently all that is necessary. If this does not suffice, the antra may be opened below the lower turbinate. This is often the only intranasal surgery indicated, it being seldom that operations on the ethmoids or sphenoids are necessary.

While not a typical example of focal infection, as generally considered, sphenoiditis, either purulent or hyperplastic, may cause irritation of the nasal ganglion, with the manifold symptoms described by Sluder. The injection of this ganglion with 5 per cent phenol in alcohol may relieve these symptoms, but the removal of the underlying cause, namely, the infection of the sphenoid sinus, seems the logical method of attack in these cases.

THE EAR.

Infections in the ear have not been as generally recognized as foci causing general diseases as have infections in the teeth, tonsils and sinuses.

Our experience in the St. Louis Children's Hospital has convinced us that infections in the middle ear and mastoid antrum in infants may be the cause of gastrointestinal disturbances, commonly described as "cholera infantum." As the development of this phase of focal infections is recent, it will be entered into in some detail.

Hartmann,[3] in 1898, called attention to the fact that infants with infections in the ear often suffered from diarrhea, vomiting and loss of weight, and that these symptoms improved after paracentesis.

Maurice Renaud,[4] of Paris, in 1921, however, seems to have been the first to have recognized the fact that this infection may involve the mastoid antrum, and that incision into the tympanic cavity is not sufficient to relieve the condition. After autopsies which had disclosed pus in the mastoid antra of 70 infants who had died of socalled "cholera infantum," he opened the mastoid antra of 10 such infants suffering from diarrhea, vomiting and loss of weight. All of these showed improvement of the gastrointestinal symptoms, but nine eventually died, and only one survived. This high mortality, he be-

lieved, was due to the fact that the operation was not performed until the infants were in extremis, and that had he operated earlier the results would have been better.

In the fall of 1923, autopsies at the St. Louis Children's Hospital disclosed pus in the mastoid antra of infants who had died of gastrointestinal disturbances not due to food poisoning but to some parentral infection, and in whom no other focus of infection was found.

At this time we did not know of the work of Renaud or that of Dean and Byfield, at the University of Iowa. This work was carried on in close cooperation with the Department of Pediatrics, under the direction of Dr. W. McKim Marriott.

In May, 1925, Dr. A. M. Alden and the writer presented a paper[5] based on mastoid operations in fifteen cases of infants under two years of age, suffering from diarrhea, vomiting and loss of weight due to parentral infections, with eight recoveries and seven deaths.

Ten of the twelve cultures in these cases showed hemolytic streptococcus growths, and it seemed that there must be some particular strain of this organism which had a special affinity for the gastrointestinal tract, but our subsequent experience did not confirm this assumption.

During the winter of 1926-27, mastoid operations were performed on forty-three infants under two years of age, thirty of whom presented marked gastrointestinal symptoms. Twenty-two of these thirty recovered and eight died.

Cultures were obtained from sixty-seven mastoids in this series of forty-three cases. Seventy-nine growths were obtained and seventeen different organisms were isolated. These different organisms showed no special relationship to the gastrointestinal symptoms, all of the more common varieties occurring both in cases with and without gastrointestinal symptoms. So that while the hemolytic streptococcus may be a special factor in the production of gastrointestinal symptoms in infants, other organisms also produce the same effect. These infants fail to gain normally in weight and suffer more or less from gastrointestinal disturbances, and in the more severe cases have marked diarrhea, vomiting, loss of weight, toxicity and a septic temperature. These symptoms are not affected by any change of diet, but after the mastoid antra are opened

the infants thrive on the same diet which they could not assimilate before the operation.

The ear, besides acting as a focus of infection, is frequently affected by foci in other parts of the body, and this should be carefully considered in the study and treatment of all cases of deafness of nonsuppurative character, whether of conducting mechanism or perceiving mechanism type, as it is generally accepted that a chronic, low grade infectious process is frequently the underlying cause, and unless this is discovered and removed we cannot hope to check the progress of the disease.

Vertigo denotes a disturbance of the vestibular apparatus, and when actual disease of the labyrinth or central nervous system is eliminated, it is usually due to a toxic irritation from some infected focus. While this focus may be in the teeth, appendix, gall bladder, female pelvic organs or prostate, it is not infrequently in the upper respiratory tract. A careful use of the vestibular tests will enable us to distinguish these cases of toxic labyrinthitis from those due to organic lesions in the labyrinth and brain, the characteristic features in these toxic cases being that all of the reactions, both to turning and caloric tests, are proportionately affected. In the early or irritative stage, they are all exaggerated, and we speak of the labyrinths as being "hypersensitive." In the later stages, when actual destruction of the labyrinthine structures occurs, these reactions are proportionately diminished; and, if total destruction of the labyrinth occurs, the reactions are abolished.

That toxemia from oronasal sepsis may accompany neurasthenia or mental unsoundness is common knowledge. Psychic disturbances in focal sepsis are striking and often disastrous.

Among the most prominent classes of disease which our experience has caused us to attribute to focal infection are the rheumatic and arthritic cases. While some of the older and more serious types do not show remarkable improvement, the general results in these cases are such as to convince us that foci of infection are an etiologic factor, and that our lack of success can be attributed to a failure to eliminate all foci, or to the existence of some other overlooked factor.

In cardiac cases, both muscular and valvular, we are convinced of the value of the elimination of foci of infection, both as a preventive and a curative measure. In these cases, close

cooperation with the internist is most essential, as they are bad operative risks and the greatest care must be exercised to have the patient in the best possible condition when any surgical work is attempted, this, together with the postoperative care, being much more important than the mere operative procedure.

In the genitourinary group: Cases of pyelitis respond as a matter of routine to the elimination of infective foci.

Hemorrhagic or glomerular nephritis is influenced most favorably by the removal of foci of infection in the throat, nose and ear.

I wish to cite briefly the cases of two brothers which illustrate this:

C. L., aged five years, entered the hospital February 24, 1927, with acute hemorrhagic nephritis, following an acute tonsillitis. The X-ray showed a cloudiness of both antra. March 5th, irrigation of both antra disclosed pus. Windows into the antra were made beneath the lower turbinates and one-fourth per cent mercurochrome instilled. March 9th, the tonsils and adenoids were removed. The patient was discharged March 12th. The urine was free from blood, albumin and casts, and has remained so up to the present time.

His brother, J. L., aged four years, developed acute tonsillitis March 15th, probably contracted by kissing Charles on his return from the hospital. On March 24th, he developed hemorrhagic nephritis with increased temperature, albumin and blood in the urine. He was sent into the hospital March 31st for removal of tonsils and adenoids, but, because of the acuteness of his condition, operation was postponed until April 29th. The operation was uneventful and the child has continued free from nephritis.

Cases of nephrosis or parenchymatous nephritis, with edema, present a less favorable picture. They are very poor operative risks and show a marked tendency to relapse. Whether this is due to the type of organism or to some other factor is a question requiring further research for its solution.

In the ocular group of diseases, commonly attributed to focal infections, the results are usually most favorable, and sometimes almost magical, as, for example, in the case of a boy of eighteen, referred to me, who was almost totally blind in one eye. An infected sphenoid and ethmoid region was

exenterated, with almost immediate restoration of sight, and he amused himself the same afternoon by watching a baseball game from the hospital window, using only the previously blind eye. When cases are referred to us by an oculist we should be most careful and thorough in our search for a focus of infection, and be willing to operate on slighter evidence of infection than we would if they were uncomplicated otolaryngologic cases.

Pulmonary Group.—Tuberculosis will only be mentioned to call attention to the possibility of infection by way of the lymphatics from the upper air passages to the apex and hilum of the lung, but there is a type of case in children which is often diagnosed as tuberculosis, especially by the X-ray, which we believe to be a nontubercular infection from the upper air passages. The citation of the following case will briefly illustrate this type:

E. H., aged 7 years. Admitted January 19, 1924, with a history of anemia, undernourishment, sore throat, bronchitis and pus in the urine. A tentative diagnosis of tuberculosis had also been made. Weight, 39 pounds; height, 42¼ inches.

The X-ray report of the chest was as follows:

(a) X-ray of chest, anteroposterior: "Much increase of hilus shadow on either side. Lung markings are thickened, nodular and extend fairly well out. Throughout the upper section on either side there is a large amount of fine soft and coarse parenchymatous mottling.

"X-ray diagnosis: Pulmonary tuberculosis."

(b) X-ray of sinuses: "There is a general haze over both antra."

"X-ray diagnosis: Bilateral maxillary sinusitis. The tuberculin skin reaction was negative."

On admission he had a bilateral acute otitis media, which promptly subsided after paracentesis.

Nose and throat examination revealed a tonsillar stump remaining on the left side, a mass of adenoid tissue in the nasopharynx. The nasal mucous membrane was swollen and covered with a thin mucoid secretion. The remains of the tonsils and adenoids were removed. Windows were made in the antra, below the lower turbinates, and one-fourth per cent mercurochrome instilled.

The nasal infection cleared up, the appetite improved and the weight increased.

Patient was seen February 2, 1925. Weight, 52½ pounds; height, 46½ inches. Urine negative; ears, nose and throat negative.

ASTHMA AND HAY FEVER.

Although there is neither time nor occasion to discuss this complicated question in detail, many cases are relieved or improved by the elimination of foci of infection in the upper air passages.

Glandular System.—The direct connection of the lymphatics of the upper air passages with the deep and superficial cervical glands accounts for most of the infections of these structures. In fact, enlargement or induration of these glands should cause us to search most diligently for a focus of infection either in the throat or the sinuses.

Infectious diseases, such as scarlet fever, diphtheria, measles, poliomyelitis, etc., will only be mentioned to call attention to the upper air passages acting as a port of entry for the general infection.

In diseases of the nervous system, we feel that chorea may be benefited by the removal of infected tonsils and the correction of other foci in the upper air passages.

Septicemia, with the port of entry in the ear, nose or throat, is much more frequent, especially in children, than is realized, unless routine blood cultures are taken. While many of these cases are unrecognized and undergo resolution spontaneously, the removal of the offending focus is the surest method of curing them.

Skin Lesions.—Under this heading the writer desires to call attention to a condition in infants which seems to have a direct relation to infected tonsils and adenoids, namely, erythema, sometimes called acrodynia, or "pink disease." This affection usually occurs in children, from three months to three years of age. The onset is insidious, with poor appetite, malnutrition, restlessness, irritability, wakefulness, profuse sweating and a leucocyte count of 10,000 to 15,000. The characteristic feature, however, is the eruption, which is macular in type, with miliary papules. This may involve the entire body, but is most marked

in the extremities. The hands and feet are red and swollen, but feel cold to the touch. Desquamation, ulceration and loss of the nails, or even the toes, may occur. The nervous symptoms are especially prominent. The persistent insomnia, the restlessness, irritability and constant rubbing or scratching of the extremities are most striking. The duration is given as months or even years.

We have recently seen several of these cases and their improvement after the removal of tonsils and adenoids has been much more prompt than the usual description would lead one to expect.

One of the most striking features of our experience with diseases due to focal infections has been that the results obtained are by far the most favorable if the offending organism is the hemolytic streptococcus.

CONCLUSIONS.

1. Focal infections are frequently the cause of general diseases.

2. A large proportion of these foci are found in the ear, nose and throat.

3. The fact that multiple infections may exist must be borne in mind and all possible foci considered.

4. Finally the writer again wishes to emphasize the statement that this problem does not consist merely in the discovery and elimination of an offending focus, but is one which requires the most careful study of the case from every angle and the closest cooperation between the various consultants.

5. While this general care and observation is essential in these cases, the prime importance of discovering and correcting any focus of infection remains, and we can safely state our belief that no patient is made worse by the elimination of foci of infection in the ear, nose and throat.

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LXXXI.

MASTOID INFECTION IN THE INFANT.*

By George Morrison Coates, A. B., M. D.,

Philadelphia.

It seems well to preface this discussion by a definition of what is meant by the title Mastoid Infection in the Infant. As some age limit for infancy must be arbitrarily set, I will consider that we mean that period between birth and the time when the ear and its accessory cavity, the antrum, has assumed to a certain extent the characteristics of the adult. This would place the age of infancy as from birth to two years of age, although many developing adult characteristics are found before this limiting period is reached, and a great change has taken place between the middle ear of a few weeks' old infant with its almost absent air space, its short, wide, patulous and horizontal eustachian tube, its reduplicated folds of attic mucosa, its obtuse angled drum setting, lack of bony external canal and mastoid process and cells, except the relatively large mastoid antrum; and the adult type structure developing rapidly as the months go on. The infantile ear that must be considered here as presenting a different question of diagnosis is the one just mentioned. It must be understood that many mastoids well below the period limit of two years show a marked development of the mastoid process with pneumatization.

What is meant by the term mastoid infection? In the adult type, undoubtedly, it means sufficient infection of the mucosa of some or all of the mastoid cells, with or without bone destruction and thrombophlebitis of the blood vessels, to give rise to local manifestations of inflammation over the mastoid area, systemic signs that can be attributed to such an infection, or infection and destruction of mucosa and bone without recognizable signs or symptoms until a long interval of time has

*Read before the American Academy of Ophthalmology and Otolarygology, September 16, 1927, Detroit, Michigan.

passed or the autopsy has disclosed the seat of disease. In the infant where the mastoid has not developed we experience greater difficulty in defining this condition, since we have, by the terms of our previous definition, no mastoid to deal with except the large antrum, or at the most a few surrounding pneumatized spaces. The mastoid is still diploic in type. Mastoiditis, therefore, to be distinguished as a separate entity from otitis media must mean infection of the antral mucosa, of the bony walls of the antrum and of such few surrounding cells as may be present, sufficient to cause local symptoms calling attention to the localized infection, or general symptoms which may or may not be linked up in diagnosis with this local infection. Can there be present in the infant's mastoid infection sufficient to cause general symptoms of a serious or fatal nature without attention being attracted to the mastoid or without the possibility, in certain cases, of making a diagnosis by observation and study of the mastoid? If so, is there a large group of such cases observable? Opinions differ widely on this matter, and as it seems a matter of some moment, it will be discussed thoughtfully and impartially later on.

With these two definitions in mind, let us examine briefly the anatomy of the infant's middle ear and mastoid. The eustachian tube is relatively shorter, broader and more patulous than in the adult; the external bony canal is lacking, the mastoid process is undeveloped, diploic in character with but one large cell, the antrum, and later a few small surrounding air spaces. The mastoid cortex is usually thin, and the mucosa of the attic and antrum redundant, lying in folds. The middle ear and antrum are normally sterile, but for the first few weeks of life, at least, the mucous membrane more or less completely fills these cavities and hearing is undeveloped until the fourth day. The inner table is usually thicker and more resistent than the cortex.

As, according to Gomperz and Alexander,[1] the infantile tympanic membrane is more resistent than in the adult, and as the folds of mucous membrane in the middle ear and attic prevent the early accumulation of a large mass of infected secretion, pressure upon the drum, with resultant necrosis and perforation, is often much delayed. This delay may be prolonged

by drainage of infected material through the wide patulous eustachian tube, so that while general symptoms of infection may be apparent, there is little to direct attention to the ear or, frequently, little to see if examination is made. Since some of the diagnostic points of mastoiditis are those of middle ear infection or its absence, the two cannot be considered entirely separately.

The ordinary frank type of mastoiditis seen at an advanced stage offers no greater difficulties of diagnosis in the infant than a similar type in the adult. There is frequently the same history of acute rhinitis (sinusitis) and tonsillitis, an attack of one of the acute exanthems, occasionally, at times, the history of trauma may be a factor. The subject is more often debilitated and undernourished than otherwise. In the youngest cases the presence of pain cannot be ascertained, and in the older ones it can only be inferred by restlessness or scratching at the ears. The fever is from moderate to high, with some irregularity of curve, and the white count may show some leucocytosis. Bacteriology in this class of case, according to Alden,[2] shows the infecting agent to be usually a pneumococcus, staphylococcus or nonhemolytic streptococcus. This is the picture of otitis media as well, for otitis media of some degree is the almost necessary precursor of mastoid infection. If the mastoid is infected, tenderness to pressure may be demonstrated by gentle bimanual pressure with the fingers, first on regions well removed from the mastoid, gradually working down to the antra. Ersner[3] strips his babies before examination, as a kicking, unclothed baby is happier, less fretful and more likely to react properly to the pressure. At the same time, the soft, velvety feel of inflamed periosteum can be noted and contrasted with the well side if the case is unilateral. Rumpling of the skin over the mastoid with the finger gives a sensation of attachment or rigidity on the affected side contrasted with free mobility on the normal. Inspection from the rear should show the auricle displaced downward and outward in an advancing case, or reveal flattening or loss of one or more of the normal postauricular creases. Frank edema is diagnostic if skin infection of the surrounding areas, including the external canal, pediculosis capitis, etc., can be excluded, and this statement applies also to the next stage—fluctuation

—which then means cortical perforation. Inspection of the external canal shows quite as constantly as in the adult the drooping of the posterosuperior canal wall, but this manifestation of mastoid periostitis is more superior than posterior because of the absence of the external bony canal and the subantral cells and the slightly higher and forward position of the antrum. The external canal, being composed solely of soft tissues, is frequently seen as a narrow slit, due, according to Alexander,[4] not entirely to swelling of the soft tissues themselves causing the posterosuperior wall to descend but also to passive stretching of the anteroinferior wall. This eliptical canal opening extends, in its long axis, in a direction from anterosuperior to posteroinferior. Often the slit is so narrow as the canal nears the drum that it will not permit the passage of a speculum, and examination of the latter is impossible. In any case, only a small speculum can be used, and a self lighted one is usually essential. The child should be rolled in a blanket and held as immovable as possible on the bed, and the canal cleansed of cerumen, pus and débris. The tympanic membrane, if seen, will now present any of the usual characteristics—inflamed, bulging in whole or in part, and red, pink or gray, perforated with discharge or approaching normalcy.

Fever, present during the preceding otitis media, may be high or absent, depending largely upon the amount of drainage present, the virulence of the infective agent and whether or not perforation of the membrana tympani or mastoid cortex has occurred. Leucocytosis of moderate degree is present, with a polymorphonuclear percentage of from 70 to 80. The X-ray gives little information, since the area involved is too small—it gives no information, as a rule, unless the child is anesthetized with a few drops of chloroform.

Perforation of the drum often occurs late in the course of the otitis media, while perforation of the antral external wall or cortex takes place easily and quickly, often with relatively few symptoms.

We must remember that pneumonias, at least of a certain type, have been attributed by creditable observers to infections in the nasal accessory sinuses or in the middle ear and

mastoid, and certainly autopsies done on victims of influenza in the army epidemic of 1918 disclosed a surprising number of middle ears and mastoids that contained purulent secretion. Was this the cause of death in those "flu" cases? Most of them during life were symptomless as regarded the sinuses and ears.

As far back as 1868, Wreden[5] observed that at autopsy of year old infants dying of bronchopneumonia, the middle ears and antra showed evidence of inflammation or infection, but attributed this condition to the pneumonia and did not regard it as the cause of the latter. These observations were confirmed by Rasch[6] (1897), who found inflammatory conditions of the ears in 42 out of 43 children having died of bronchopneumonia, and Ponfick[7] reported the same results in 10 out of 11 autopsies. This situation is attributed by Friedrich,[8] Gomperz.[9] Alexander and others to the fact that the undeveloped ear of infancy is especially prone to infection in any disorder which reduces the individual's normal resistance and interferes with nutrition, and they include in this category malnutrition, pneumonia, various intestinal affections, rachitis, tuberculosis, syphilis and others. Friedrich suggests, in 1900, that this process may be reversed and that the ear infection may be primary and the systemic manifestations secondary.

This suggests the consideration of what must be considered an atypical type of middle ear and mastoid infection in infants, the diagnosis, at least as far as the indications for operation go, depending largely on systemic or focal reactions in other parts of the body and to a lesser extent on local manifestations. As I have just pointed out, this subject and this controversy is not new, but it has recently been brought again before the profession by the reports of Byfield,[10] Marriott[11] and Jeans,[12] pediatricians, Dean, Alden,[13] Lyman,[13] McMahon[14] and others, otologists, who have advocated closer study of certain types of infantile gastrointestinal disturbances in relation to the ear as a cause of the infection. Hartmann,[15] in 1894, called attention to the significance of middle ear and mastoid infection in nutritional disturbances of infants, and Goeppert[16] believed that the otitis was secondary to the intestinal disorder and found that ear complications were so prevalent in this class that only 12 per cent of those admitted to hospital had

healthy ears. Friedrich believed that much of this ear disease was secondary to intestinal disease and loss of resistance, but says "secondary nutrition disturbances play an important rôle in primary ear disease in the case of infants. A regular digestion and a uniform increase in the body weight afford the best criterion of an infant's health, for its digestive organs are so sensitive that the slightest local or general disturbance may suffice to upset its stomach. If there is a purulent focus anywhere in the body, autointoxication will be much more likely to result from the absorption of the products of metabolism in the infant than in the adult, and will show itself chiefly in the organs which are functionally the most important—that is, it will be followed by indigestion and a falling off in weight." Hartmann[17] and Ponfick, in the Berliner Kinderklinik, found that often intestinal disturbances of an obstinate nature yielded promptly to the discovery and evacuation of infection in the middle ear.

Recent interest in this question was stimulated by the often quoted report of Renaud,[18] who in a series of 70 consecutive autopsies on infants found extensive suppuration of the internal ear and petrous bone in all, and in a large number such infection was bilateral. The histories of these cases did not show ear involvement, but they had been, for the most part, weak, undernourished children, from poor hygienic surroundings who showed, on admission to the hospital, a more or less acute syndrome manifestation by emaciation, signs of infectious intoxication, fever, vomiting and diarrhea. No significant lesions were found in the intestinal tract. The spleen, lungs, liver and kidneys all showed signs of inflammatory reactions, and there was considerable edema and congestion of the vessels in the brain, hemorrhage and thrombi being found in some cases. He thinks that the diagnosis of gastroenteritis was never confirmed at autopsy and that suppuration of the ear is the essential, if not the exclusive, factor in infant mortality.

Leroux[19] thinks that a latent otitis is the rule in every newborn infant and that the ear may be infected from the first inhalation. Kistler,[20] working in the Virchow Pathologic Institute years ago, noted the fact that in a large number of infants the mastoids were found full of pus in nearly every case. The history of these and the cause of death is not reported.

In view of these and many similar reports, the question at once arises whether these accumulations in the ear cavities are antemortem or postmortem in origin, and opinions will be found differing as widely on this subject as on all other points at issue. Alexander states that "after death the mucous tissue undergoes exceedingly rapid decomposition from putrefaction filling the tympanic cavities of infants with a gelatinous, more or less purulent fluid." On the other hand, McMahon, studying histopathologically the tissue removed during antrum drainage operations done by Lyman for the relief of gastrointestinal disorders, finds ample antemortem evidence of pathology, and Alden, Lyman and many others attest to the certainty of infection by cultural methods. This is the only way to prove the point—the mere presence of a purulent looking material at autopsy (possibly long delayed) is not conclusive.

The general concensus of opinion seems to be that middle ear (and mastoid) infection, from whatever cause, whether latent, masked or manifest, is of very frequent occurrence in infants, particularly in those with lowered resistance from chronic or acute disease, general malnutrition and unhealthy surroundings. Gomperz attributes this to:

1. The short, wide eustachian tube and the position of the nasopharyngeal opening.
2. Changes in circulation following birth, together with hyperemia and loosening of the tympanic mucosa.
3. Persistence of the embryonal tissue of the middle ear mucosa beyond the fetal period (this tissue being especially subject to infection).
4. Immaturity and undeveloped resistance.
5. Heredity and constitutional taints—tuberculosis, syphilis, rickets.
6. Infectious diseases—sneezing, crying, coughing and vomiting forcing infection into the ear through the wide, short tube.
7. Hematogenous infection.
8. Gastrointestinal and cerebral affections in which vomiting exists.

The tympanic cavity of infants contains abundant mucous tissue and fluid, according to Alexander, which makes an excellent culture medium for invading bacteria. This myxoma-

tous tissue, embryonal in type, lies in many redundant folds, in great quantity, and is subject to rapid purulent transformation. The accumulation of this tissue, with connective tissue bridges, fills the epitympanum and much of the mesotympanum. The folds are thick and succulent, and when invaded by infection there is not at once one large cavity containing pus but many small ones communicating with difficulty, rarely causing perforation of the drum during the early stages for this reason, but offering ample chance for absorption by lymph and blood vessels, and to a lesser extent discharge into the stomach and intestines through the wide eustachian tube. Bryant[21] states that the lymphatics are nine times more permeable than in the adult, and therefore nine times more capable of transmitting the products of infection. The mucosa of the mastoid antrum and additus conforms in general to that of the middle ear, and closure of the aditus by swelling of infected mucosa may easily take place and may well leave infection trapped in the antrum after the infection of the tympanic cavity has subsided. Absorption from such a latent focus may readily be accountable for focal symptoms with little evidence of local trouble.

Wide differences of opinion exist among both pediatrists and otologists as to the cause and effect relationship of gastrointestinal affections and otitis, just as they existed thirty or forty years ago. Possibly the solution is nearer at hand than many think.

It is in this class of cases, namely, middle ear and mastoid infection associated with systemic manifestations in other parts of the body, either in the respiratory or gastrointestinal tracts, that the present day problem of diagnosis is difficult. What is this association—cause or effect, post hoc or propter hoc? The recent notable work of Alden, Lyman, Dean, Byfield, Marriott and Jeans in the West has caused great renewal of interest in this question, and in some instances alarm, less overenthusiasm lead to indiscriminate operation without due and careful study and diagnosis in the individual case. And opinion seems to vary sharply, depending on geographical location. When these eminent men tell us that they believe the socalled cholera infantum syndrome, and other intestinal manifestations, are caused largely by tympanic infection, great respect must be paid to their opinions, since they are reinforced by

many detailed case reports showing gain in weight, lowering of fever and cessation of diarrhea when the middle ear or mastoid antrum is drained. They have seen many such cases in a relatively short space of time, and in consequence the mortality from these affections has been reduced, by their method of treatment, to a marked degree, many of these cases formerly being considered hopeless.

On the other hand, Eastern pediatricians tell me that, since the rigid municipal supervision of the milk supply and general improvement in the hygienic and sanitary conditions of the home, very few such cases are seen by them, either in private or hospital practice. The chief of pediatric service in the largest hospital in Philadelphia tells me that he sees but one or two such serious cases in a year, whereas formerly he saw many. A leading New York pediatrician and author says the same thing, and that his cases of malnutrition, athrepsia, dehydration, etc., yield to nonotologic management. The opinion of these men is also entitled to respect. Is it possible that we are dealing with and talking about similar conditions with a different etiology? Jeans (quoted by Dean) has suggested that this might be so, and that the climate of Iowa might be responsible for the apparently greater prevalence of such infections there.

Otologists, also, in other parts of the country have been rather inclined to look askance on the mid-west practice, being influenced by their pediatricians and seeing few cases, although few otologists of experience anywhere cannot recall gastrointestinal cases, or cases with fever and malnutrition of obscure origin, that cleared up promptly after a myringotomy on an almost normal appearing drumhead. Such instances must give us pause, but that a bilateral mastoid operation should be performed by the otologist on apparently normal ears, on the simple demand of the pediatrist, as I have heard recommended in open meeting is, I think, dangerous to the reputations of both specialties, to say nothing of the welfare of the patients.

Is it necessary or practical then, for the pediatrician to make the diagnosis for the otologist? What are the otologic findings that can be considered diagnostic in these cases of atypical or masked infantile mastoiditis? If there is aural discharge, a

bulging drum, mastoid edema, etc., the case comes under the heading discussed at the beginning of this paper. No one questions the necessity of a drainage operation under these circumstances. Aside from these, the trained otologist, examining carefully with brilliant light in a magnifying otoscope, will find certain guiding signs of infection if it is sufficient to cause the syndrome. Slight discoloration of the drum, a change to a grayish color with loss of luster, giving it a ground glass appearance; slight fullness, or redness, particularly of Shrapnel's membrane, slight posterosuperior canal wall sagging—these are all that may be found. Alden states that the infecting organism in these cases is almost invariably the streptococcus hemolyticus. Are these enough to enable us to make an operative diagnosis? Yes, when combined with continued fever, loss of weight, dehydration, diarrhea and vomiting, otherwise unexplainable; no, decidedly, when these or similar general symptoms are not present. To say that every case of infantile diarrhea, with slight fever of but short duration, should have a double mastoid drainage in spite of the absence of all otologic signs, is, of course, reductio ad absurdum; and yet that is precisely the attitude that is being held and discussed by pediatricians and otologists in certain portions of the country. Those holding this view need the curb; possibly also those who are entirely opposed need the spur to seek more carefully for otologic reasons for the few cases that they report seeing. No one will dispute the dictum that every infant, with or without some signs pointing definitely to the ears, who has fever, diarrhea lasting more than a day or so, any nutritional disturbance or respiratory symptoms should have a careful and thorough ear examination. If ear infection is found, the prompt institution of ample drainage is good practice, and if a myringotomy with free drainage fails to relieve the situation, postauricular drainage is indicated, for copious, prolonged, increasing, or at least nondiminishing aural discharge is in itself indicative of mastoid involvement.

Although the question of operation, whether myringotomy or antrotomy for drainage, is somewhat outside the scope of this paper, it has seemed to me that in grave cases operation adds little to the hazards of the case. I have for years taught my students that in case of doubt it is better to drain where

systemic symptoms are grave, even when local ear manifestations are inconclusive; and we have all seen striking results from this procedure, which apparently demonstrate that the ear infection is, at least in many cases, primary. We should, however, subject our cases to the closest possible study and scrutiny, and be constantly on the watch for means of improving our methods of otologic diagnosis in these cases.

There are then a number of hypotheses open for consideration in these cases of systemic infection with apparent mastoid involvement.

1. Is the condition of the mastoid found at autopsy in the series quoted due to postmortem changes?

2. Is the middle ear and mastoid infection secondary to the systemic infection, and if so, is it due to a consequent lowered resistance or to forcible injection of infected material through the eustachian tube, or to both?

3. Are there different types of cases of different etiology with serious gastrointestinal symptoms in different parts of the country?

4. If serious or fatal gastroenteritis is caused by middle ear and mastoid disease, should not cases of infantile pneumonia and bronchitis be given equally careful study and examination with the expectation of discovering a similar etiology?

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LXXXII.

A SERMON.*

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"Age cannot wither nor custom stale their infinite variety."

This discourse will refer to no Shakespearean heroine of the Nile, but rather to certain operations as now practiced upon one of the accessory sinuses of the nose and incidentally to certain human emotions and their phases which directly concern the doctor of laryngology, particularly in his relations to himself, his colleagues and incidentally to the laity at large.

Speaking of operations, did it ever occur to you that there is no standard or set procedure for certain definite pathologic entities, so to speak, that may occur in these cavities. It is true that in the event of an ethmoid suppuration we have a number of methods immediately at hand, such as the Hajek, Mosher, Sluder, Ballinger, etc., etc., but which one is indicated for that particular case? Again, a frontal sinus may present itself requiring some form of an external operation. What method is applied? If the patient happened to be in Germany, a Killian would unhesitatingly be performed; if in France, some modification of the Luc; if in England, one of the methods advocated by Tilley or Watson Williams, while here one of these foreign procedures as modified by that particular operator. The only purely original American method that we have appears to be one devised by a general surgeon, Lothrop, and as far as I know is only used in a modified form by the American sinuologist. Now how did all this come about? Why do we not have a systematized procedure in the application of surgical technic in the sinuses as well as, we will say, in the upper abdomen? I think that this can be answered by contemplating the lack of uniform teaching in the postgraduate schools

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and courses in the United States. Each individual teacher here has usually received his training from one or more European clinics and is naturally prone to follow the teachings of a certain particular outstanding authority who had always been a hero in his eyes. This follows that his students in turn will be schooled in the belief of that particular Hofrat or professor and therefore will have a decided English, German or French leaning, as the case may be. Now, in passing, this brings the thought of our relations to foreign countries, notably the German speaking ones, as far as our specialty is concerned. Gentlemen, the time has passed when we were subservient to the teachings and methods which had their emanation in these sources. A pilgrimage to these shrines lasting but a few weeks, compared to the available, well rounded postgraduate courses of some of our own universities, is like sending a boy to a man's work—he can attempt it, but what he accomplishes is only a smattering and usually badly done at that. Reverting again to the lack of uniformity in sinus technic, other influences besides teaching have crept in, such as custom, precedence, ability, ignorance and even intolerance. Regarding custom, precedence has been all too slavishly followed. Suppose we continued the teachings of our predecessors in their originality, how absurd certain things would seem. For instance, Charles II had a law enacted which prohibited coaches from the streets of London on account of the destruction caused by their wheels to the paving stones. So do the trucks nowadays, but instead of abolishing the trucks we concrete the roads. Again, coming down to our own time, who does not remember when everybody drank from the same cup at the public fountain, when we rode in street cars pulled by horses, and nobody took a bath until Saturday night? But you say all this has changed in the scientific light of a new era. Very well, let us see. Since the origin of the species, the prehistoric man meeting with another and not wishing to fight dropped his weapon and extended his right hand as a symbol of friendliness. This original purpose has long since passed into forgetfulness, but it is still the custom to shake hands on meeting a friend or acquaintance. Another instance: In the dark ages medicine and magic were almost synonymous. When a patient consulted the doctor-magician, it was a custom for the medical man to place his

lips on the part that was ailing and suck out the evil. We all know how to this day a bruised finger is instantly placed in the mouth and sucked, and the old nursery remedy of kissing it and making it well no doubt descended from this same origin. Scratch the epiderm of the civilized man and the barbarian is found in the derm.

Now let me cite another example in close relation to medicine. Erysipelas, which still sometimes occurs as a complication of purulent sinusitis, but less and less in evidence owing to better asepsis in our methods of treatment. Formerly it was considered not only a complication but an etiologic factor of no little importance, and even Hajek in a recent edition of his well known monograph mentions it with more than passing interest. Here is another illustration of the changing order of things, for as late as the seventeenth century St. Anthony's fire took its toll of victims, were they prince or pauper. It is curious to recall that this disease received its name of St. Anthony's fire because sufferers from it were apt to be cured after being treated by the wise monks at the Abbey of St. Antione at Vienna. St. Anthony of Padua had nothing to do with it as is so commonly believed.

Again, common table salt would seem to be the last substance in the world around which one would associate romance or superstition, and yet this substance has been held in the highest esteem since the prehistoric era. The Israelites at the time of Moses did not believe proper digestion could occur unless salt were taken with their meals. At one time, during the Graeco-Roman period, officials and employees of the State received a portion of their pay in salt, which was termed salarium, from which our word salary sprang. In medicine the application of salt to a wound or sore is still in vogue, and even its accidental spilling has long since been accounted an omen of ill luck, originating perhaps in the tradition that at the Last Supper Judas, while in the act of presenting salt to the Master, spilled some upon the table. An old saying has it, "When a man once comes to believe a thing he is loath to discredit it," and habits are even more enduring than beliefs.

Let us now consider our discourse from the standpoint of personal ability. Some of us are blessed with a superabundance

of natural ability, but most of us have to acquire it through continued practice, observation and personal contact with men noted for their skill along given lines. I, of course, refer to operations and operative technic. The first requisite for a given operation is proper and suitable instruments. I recall the first course I attended in operative work on the sinuses of a cadaver in Vienna, where the only instruments available were those ordinarily seen in the deadhouse. Naturally the operations, like Gay-Lussac's law governing gases, were directly as to the conditions of the instruments. One cannot make pancakes without batter, neither can one perform an operation properly without suitable tools. For this very reason it is proper that we, in the beginning, select certain instruments that appeal to us and practice with them (preferably on the cadaver) until their feel becomes almost of a second nature. How many of us visiting clinics in other cities and noting with what ease a certain man performed a difficult operation, have bought his instruments and at the first opportunity, after arriving home, tried them out with results that were anything but pleasing. Now this miscarriage of technic did not result because operator A was necessarily more skillful than operator B, but because B did not have the same mastery of the procedure as A, having never before applied it. The chances are that A in the beginning had quite as much trouble as B, and only by constant study and application was he able to master it to such a degree as to make a seemingly difficult method look comparatively easy. So while there is a certain beneficent aspect toward the unusual and spectacular, nevertheless it is, after all, the end results that count, so that if operator B, by his method procures the same result as operator A by his novel but more or less involved procedure, there exists no reason (other things being equal) for one to claim superiority over the other, both in the final analysis having been to a like degree successful. And yet we cannot assume this attitude to such a degree that we become intolerant of another's views or methods. Intolerance has played a continuous rôle since the early dawn of history. Moses was denounced and threatened with violence when he first tried to free his people from Egyptian slavery. Socrates, for his philosophic truths, was given the cup of lethal hemlock, and you all know what happened to the

Man of Galilee when He preached peace and good will to all men: He was taken out and nailed to a cross. Intolerance has no place in medicine and never should have had, although in the middle ages our profession subsisted upon it, traces of which remain to the present day. When new theories are advanced or new procedures recommended, many of us are prone to give heed with half an ear, so that when asked an opinion regarding them are apt to reply that we do not think much of it, or that we do not do this form of operation. The world little cares what we don't think or what we can't do, but the world, particularly the medical profession at large, is vitally interested in what we do think and what we can do. Respecting this, how much better it is to be a positive and constructive man, everyone knowing where he stands, than a negative or indifferent one, whose beliefs and tenets are unconvincing and whose actions leave much to be desired.

Collective certitudes are infinitely superior to individual platitudes. This applies to operations as well as observations. An operative procedure that has received the stamp of approval collectively, so to speak, is worth a hundred methods that have only the support of single individuals, mostly their originators, to recommend them.

Now a word regarding the intranasal operations under local anesthesia, particularly those relating to the ethmoid. I have always been a rabid advocate of spending all the time necessary in procuring a thorough anesthesia of the parts before making the first incision. From my observations in many clinics, in perhaps as many different lands, I have long since been convinced of the great wisdom of this principle. In the first place, while you have assured the patient that there will be no pain, nevertheless if he is human he has not been entirely convinced and is apt to be a bit "jumpy" until he finds that after your thorough anesthetization the attacks upon his bulla or middle turbinate, as the case may be, are really painless. He will then be possessed of an assurance, and the operation will usually be concluded in peace and quiet. If, however, the cocainization is insufficient or but partially covers the operative area and we have an apprehensive, neurotic individual to deal with, at the first stroke of the knife there will be much noise,

some confusion and nothing accomplished. The more thorough the preparation of the parts the more quiet the patient and the more successful the operation. Show me a case in which the anesthesia has been imperfect, and I will show you an imperfect operation. This adage may not hold good in every case, but if you will take the trouble to make a thorough examination where it has occurred, even if it is one of your own cases, I think you will bear me out in this assertion. Now, coming to the operation itself: If when contemplating the advisability of an operation in a certain obscure case the thought comes to us, "That which the fountain sends forth returns again to the fountain," it may very materially change our viewpoint in that particular case. To deliberately rip out an ethmoid capsule for the purpose of removing the source of an unlocated suppuration in the nose is like putting one's hand into a basket of snakes to find an eel. You may be successful, but the chances are small; furthermore. you may get your hand badly bitten into the bargain. Even though we find to our own satisfaction that pus is continually coming from a certain portion of the ethmoid, constantly appearing in the same place after wiping away, we must not think of it as an abscess, but rather as a product and not the actual cause of the disorder, so that the mere evacuation will have little influence upon the trend of the infection unless adequate aeration and drainage is at the same time effected. Regarding the operation itself, I am inclined more and more toward conservatism, especially in those cases where little surgery has as yet been attempted. The removal of the middle turbinate, with daily applications of a strong colloidal silver solution on tampons placed up and against the ethmoid capsule, which are permitted to remain for several hours, will ofttimes bring order out of chaos and obviate the necessity of further surgical procedures. If, instead, we should meet with one of those cases which has undergone repeated operations and the entire ethmoid capsule is one disorganized suppurating mass, of course, only a complete exenteration beginning with the uncinate process will suffice. I am inclined, however, to feel toward the socalled complete exenteration of the ethmoid as Voltaire said regarding the Holy Roman Empire, namely, that it was neither Holy nor Roman, nor was it yet an Empire, so that the complete intra-

nasal exenteration, while indeed internal, is neither complete nor is it an exenteration.

And now, in conclusion, a word to our younger brethren, although to some of us older ones it might also not come amiss, and that is sticking to our ideals. Every man that reads medicine must sooner or later come to the conclusion that he is about to embark on a career that is a profession and not a business, a career in which the ideals are diametrically opposite to the accepted tenets of modern business. In the profession, the end results concern themselves primarily with the final condition of the patient, and secondarily with the remuneration received while in business. The final analysis rests entirely upon the credit side of the ledger, regardless of the fate of the house with which the trading has been done.

The younger man, starting out with ideals along these lines, will soon find himself sorely beset, not only from those who are openly hostile, but from sources which he feels have only his best interests at heart. I recall only too well, some twenty-odd years ago, a young man, having spent nearly two years abroad, returned from Vienna full of enthusiasm and with what he thought a message. This message was the recognition and treatment of accessory sinus disease. After much preparation and forethought, the evening came and the message was delivered. Instead of a lively discussion following, it was received in cold silence, and after the meeting had adjourned one of the older men approached the essayist and, putting a warning hand on his shoulder, said, "My boy, if you don't stop sticking those needles into people's antrums you are going to kill someone." This was most disquieting to the young man and far from encouraging him in further investigations along sinus lines; but, thank God, he had the courage of his convictions, and now is before you today, ready to stand or fall with those same implicit beliefs.

And now, gentlemen, I am done. I appreciate this presentation has been rambling and often far afield from the province of laryngology, or for that matter, even medicine, but its intention has been largely for the ears of our younger brethren. We must always look forward, not as the Chinese do—back-

ward with their ancestor worship, else we remain as they have, stationary for the last five thousand years—but with the spirit of progress guiding our thoughts, we can revert to the Chinese and their serenity of purpose, gleaning much from their psychology, and when beset with discouragement and doubts we can indeed say, with our celestial friends, "Even though the wolves may howl, the moon shines serenely on."

LXXXIII.

THE INFLUENCE OF PARANASAL SINUS INFECTIONS IN INFANTS AND YOUNG CHILDREN UPON CERTAIN SYSTEMIC CONDITIONS AND THE INFLUENCE OF CERTAIN SYSTEMIC CONDITIONS IN INFANTS AND YOUNG CHILDREN UPON THE METHOD OF TREATING COEXISTING SINUSITIS.*

By L. W. Dean, M. D.,

Iowa City.

In 1918, before this association, I[1] reviewed the literature on paranasal sinus infection in infants and young children, and called attention to the frequency of its occurrence, and expressed the opinion that it was a common source of infection. In 1919, Dr. Armstrong and I[2] discussed the bacteriology of sinus disease in infants and described the production of experimental arthritis in animals by the inoculation of organisms isolated from the sinuses. In 1920, I[3] called attention to the occasional necessity of doing a radical operation on the paranasal sinuses of children because of systemic complications.

In 1921, Byfield[4] discussed the systemic complications that were studied at that time, and I[4] described the method of diagnosis. In 1923, I[5] reviewed the complications of paranasal sinus disease in infants and young children. Arbuckle[6] in the same year emphasized the importance of paranasal sinus infections in children and spoke of the gratifying results secured from treatment. Marriott and Clausen,[7] also in 1923, reported that acute parenchymatous nephritis was influenced favorably by the drainage of the nasal sinuses. Clausen[8] considered sinus disease to be an important factor in the causation of this condition.

Byfield,[9] in Abt's Pediatrics, discussed the systemic effects of infections of the upper respiratory tract. In the same

*Read before the American Laryngological Association 1927.

work I[10] elaborated upon the diseases of the paranasal sinuses in infants and young children.

Jeans,[11] before this association, cited a series of remote effects of sinusitis in infants and young children. Marriott[12] at the same meeting discussed the relationship between nephrosis and hemorrhagic nephritis in infants and infections of the upper respiratory tract. Shea[13] has discussed the relationship between these infections and systemic disease. Marriott[14] is of the opinion that the first step in the successful treatment of parenchymatous nephritis is to locate and treat the infection responsible. In 1925, I[15] discussed the significance of the pathology of sinus infection in infants and young children as it relates to systemic disease.

Byfield,[16] Floyd,[17] Marriott,[18] and Jeans[19] mention the symptoms of acute otitis, especially those that are referred to the gastrointestinal tract, dehydration, diarrhea, loss of weight and food refusal, and Jeans[20] reported that the same clinical picture of cholera infantum or alimentary intoxication, with minor differences, may be produced by sinus infection in infants under two years of age.

The influence of systemic conditions on the treatment of acute otitis I[21] have recently discussed.

The following conditions in infants and young children may result from a focus of infection: cardiac lesions, rheumatic fever, chorea, nephritis, pyelitis, certain cases of cyclic vomiting, deforming periarthritis, anemia, anorexia, malnutrition and chronic digestive disturbances. The focus of infection may be in a paranasal sinus.

Remote effects more or less peculiar to paranasal sinus disease are bronchiectasis, asthma and the cholera infantum symptom complex.

The presence of one of these systemic complications of paranasal sinus disease immediately influences the treatment of the infection. It is much more important to eradicate the sinus infection than if the complication did not exist. Frequently the condition necessitates immediate control of the sinus infection, as, for instance, in cases of dehydration, diarrhea and malnutrition in infants. It often indicates the need of eradication, if possible, of the last vestige of the sinus infection, as

in heart lesions and arthritis. Again it may indicate the need of conservative treatment of the sinus disease, as in parenchymatous nephritis. Patients with parenchymatous nephritis are very susceptible to infection. In my judgment, drainage of the sinuses by operative procedure should be postponed until the nephrosis is controlled, if possible, because of the great danger of septicemia following minor operations in this condition. The presence of nephrosis indicates the need of careful examination of the paranasal sinuses, and if sinusitis is found, the complete eradication of it at the proper time is indicated. Clausen[22] in a recent article shows the marked influence of treatment of the sinuses in nephrosis with edema and albuminuria. Immediately following the treatment there was a rapid disappearance of the edema and albuminuria and a rapid increase in the actual weight of the child. Previous removal of faucial and pharyngeal tonsils had not produced a favorable result.

R. P., aged eight, had a pediatric diagnosis of nephrosis. The laryngologic diagnosis was chronic paranasal sinus disease. This child was very critically ill, and only the mildest form of treatment was used. We used 3 per cent ephedrin in the nose, waited three minutes until the mucosa had shrunken and then applied the ephedrin again, with the expectation that there would be a maximum dilatation of the ostia of the sinus. After complete shrinkage of the mucosa, argyrol or some similar drug was used. This patient was pursuing a favorable course, when on March 13th he took cold, and his temperature went as high as 105°. More energetic treatment with ephedrin resulted in the control of the fever without using any treatment which would traumatize the mucosa of the nose.

M. C., aged 5 years, had a combination of glomerular and tubular nephritis with chronic suppurative paranasal sinus disease. In this patient, as in the previous one, the sinus infection was apparently controlled nicely by the use of ephedrin. At one time this patient had two spikes of temperature which both the pediatrist and the laryngologist thought were due to a blocked sinus. At any rate, ephedrin was used more persistently and the fever disappeared. Sinus disease, if chronic, is not, as a rule, eradicated by the use of ephedrin, argyrol, etc.,

but in infants and children it can frequently be controlled until the patient's condition is such as to allow of surgical treatment.

According to Jeans,[20] paranasal sinus infection may cause the cholera infantum syndrome, with minor differences, the same as acute otitis. Baby J., aged one month, had a pediatric diagnosis of anhydremia and diarrhea, with loss of weight. The otolaryngologic diagnosis was acute paranasal sinus infection. Following treatment of the nasal sinuses the same improvement occurred in the infant's condition as has been many times noted following myringotomy or mastoidectomy for otitis media: the stools decreased, there was a rapid increase in weight and the temperature curve became flattened. If the pediatrist refers to the otolaryngologist an infant with the cholera infantum syndrome, we should not stop with looking at the ears but should also examine the paranasal sinuses, and if they are infected they must be treated. Also, other acute infections about the nose and throat must be looked for. The common condition found with this syndrome is acute otitis plus paranasal sinus disease.

While all acute infections of the nasal sinuses can be eradicated by simple treatment, as the use of ephedrin or argyrol in the nose, diet, hygienic conditions, etc., frequently the control is not rapid enough to meet the situation. If with this conservative treatment of the acute paranasal sinus infection the pediatrist feels that the infant is becoming worse instead of better and there is no other source of infection, if the maxillary sinuses are involved they should be washed out. This can be done nicely under ethylen anesthesia with no harm to the patient.

Baby M. W., aged 17 months, has a pediatric diagnosis of dehydration with loss of weight. There was a fever of 101° but no diarrhea. The otolaryngologic diagnosis was an acute infection of the nasal sinuses. The nose was treated with ephedrin and argyrol. Immediately following the treatment of the nasal sinuses the temperature curve flattened and there was an increase in weight.

The difficulty in treating infants with diarrhea, dehydration, fever, food refusal, loss of weight and prostration is that we usually have an acute otitis and acute paranasal sinus disease

present. Many of these babies are so ill that it is quite impossible to drain both the ears and the nasal sinuses without producing a fatality, and it is only after careful consultation between the pediatrist and the otolaryngologist that the best method of treatment is chosen. As a rule, I think we are safe in doing myringotomy or mastoidectomy as seems necessary for the acute otitis and treating the acute paranasal sinus disease conservatively. The finding of very severe and acute infection of the paranasal sinuses at autopsy in some of the infants so treated makes one somewhat doubtful as to the correctness of this procedure. I am inclined to believe that if on the third day following active ear treatment by mastoidectomy, if the otitis is progressing favorably, the paranasal sinus infection which is present should be attacked energetically. Local treatment of any paranasal sinus disease present is always indicated. In a number of infants this has given us very satisfactory results. Occasionally, after consultation with the pediatrist, it may be advisable to drain both the ears and a sinus immediately and at the same time.

Baby L. W., aged two years, was in the pediatric service with a diagnosis of diarrhea, fever and loss of weight. Nine hundred and fifty grams were lost in four days. The baby had an acute right otitis and an acute right maxillary sinusitis. The baby was very ill and the pediatrist advised thorough drainage. Because only one ear was infected and one sinus, we drained both the maxillary sinus and the mastoid at the same time. The baby made rapid improvement and the result was very satisfactory.

Sometimes the history of the case suggests that the sinus infection is the more important one. In one instance the malnutrition began following a head cold six weeks before the infant was seen here, and the severity of the malnutrition varied with the severity of the nasal infection. The infant also had an acute otitis. In this case the sinus trouble was treated alone with marked improvement. Later the acute otitis was attacked. Finally, after the disappearance of the otitis, a satisfactory result was secured only by a thorough elimination of the paranasal sinus infection. In this instance the predominating cause of the malnutrition seemed to be the infection of the sinuses.

Infants with malnutrition, food refusals, diarrhea, dehydration, fever or prostration, or with several of these conditions together, may suffer from acute otitis, acute sinusitis, acute tonsillitis or acute retropharyngeal abscess. If any of these conditions are present the pediatrist should know of them. They should be treated. If these conditions are not diagnosed, exploratory operations on the mastoids or sinuses are not justified.

In treating infants and young children there must be the closest contact between the pediatrist and the laryngologist. Personally, I have more confidence in the opinion of the pediatrist as to the need of more energetic treatment of the paranasal sinus infection than that of the laryngologist. The pediatrist is the best judge of the seriousness of the case and of the probable source of the trouble. He is not the best judge as to what otolaryngologic conditions are present and the results that are to be secured by the various types of treatment.

In handling the serious acute infections, I work always with the pediatrist on one side of the infant and myself on the other. There should be complete confidence in each other and intense cooperation. After all the information has been furnished by each, the best judgment as to what should be done can be reached.

No rules that are infallible can be laid down for the treatment of paranasal sinusitis. In the more chronic conditions the laryngologist must keep in touch with the pediatrist and be guided by his observation regarding the improvements in the systemic condition. It has happened in one case of arthritis in a child, that the pediatrist has sent the child back three times with a statement that we have overlooked an infection about the nose or throat before the foci were eradicated and a beneficial result secured. If a patient is in our service with a chronic complication of sinus trouble, we always, even with apparently the very best result, have sent the patient back to the pediatrist with the request that he be returned again to us if there is any evidence that a focus of infection still exists.

The same relationship between arthritis and sinusitis may exist in young children as in the adult. Dr. Jeans, before this association in 1925, said that the improvement in arthritis following the treatment of sinusitis in many instances has been

so definite and marked as to be convincing as to the rôle this infection has played. To see children with deforming arthritis associated with severe pain and fever become comfortable and afebrile, and to see functions gradually restored as the result of treatment of the sinuses is impressive, especially when in former years one has been accustomed to see all efforts unavailing and the disease becoming progressively worse.

Baby E. T., aged three years, was referred to us because of chronic multiple infectious arthritis. Following the removal of the faucial tonsils and adenoids, the arthritis improved but persisted. Six months later the maxillary sinusitis was treated and the arthritis disappeared and with it the joint trouble. Two years later the first recurrence of arthritis occurred following a severe cold. The maxillary sinusitis was again treated and the arthritis disappeared. The sinus disease was permanently eradicated, and there has been no recurrence of the arthritis to date. The last treatment was in July, 1920. The report of this one case might give the impression that the treatment is a very simple matter. Unfortunately, in arthritis as in bronchiectasis, chorea and asthma cases, it is very difficult to sufficiently eradicate the chronic paranasal sinus infection to obliterate the focus about the nose.

In 1925, I[15] showed before the American Laryngological, Rhinological and Otological Society a series of pathologic specimens. The pathology of sinusitis as demonstrated at that time is such that with the very best of operative work the infection may persist. There is present in chronic sinusitis in infants and young children, the same as in the adult, subperiosteal abscesses and abscesses of bone, which frequently are so extensive that they may persist in spite of all treatment.

In the four systemic conditions above referred to, it is occasionally necessary to operate the ethmoid sinuses, even in children. After months of work on the sinuses I have seen asthma, arthritis and bronchiectasis improve after ethmoid operations. The ethmoids that we have operated upon were mere masses of softened necrotic bone, and it is inconceivable that these could ever be of any possible value to the individual. Every time I have operated upon the ethmoids the result has justified the procedure.

Every week in our service it is necessary for us to tell the orthopedic surgeon and the pediatrist that we have eradicated the manifest paranasal sinus infection, but that if they feel at any time that a focus is still present, the patient should be returned for reexamination, and these patients often do come back with a reddened pharynx suggestive of persistent sinus infection, and after a continued search for the infection I have always discovered it. I, however, have not always been able to obliterate it.

Many of these cases are very distressing. Some of the children have every joint ankylosed. The neck is rigid and there is ankylosis of the mandible. They cannot move hand or foot. We find chronic suppurative sinusitis with bone involvement. In many instances the purulent discharge persists in spite of all my efforts. The more severe and extensive the arthritis in children, the more chronic and difficult to heal is the sinus disease.

Our patients are almost all indigent cases. The choice of treatment in chronic paranasal sinus infection in young children is diet and proper climate. With our indigent cases our progress in midwinter is very discouraging. In summer the results are much better. Unfortunately, the recurrences during the winter are numerous, and with the recurrence of the sinusitis we have recurrence of the systemic condition. Given a case of arthritis, especially in which there is a relationship between the arthritis and a head cold, I would make every effort to obliterate even a mild chronic sinus infection. In the absence of arthritis but little attention would be given to it. In short, arthritis urges us to persistent careful examination and to operative procedures which are sometimes so radical that we are sometimes disturbed by the treatment instituted.

In bronchiectasis in children, the treatment of the coexisting chronic suppurative pansinusitis has given us our best results. More good has been secured by such treatment with proper diet than by bronchoscopic treatment, posture, etc. I note that occasionally the desired favorable result was only secured by operating upon the ethmoid labyrinth. In some cases work on the maxillary sinuses alone has given very satisfactory results.

H. B., aged six years, had a diagnosis of bronchiectasis. Treatment of the chronic suppurative pansinusitis which was present with ephedrin and twice a week, at first, the washing out of the maxillary sinuses and later once a week, resulted in the apparent eradication of the sinus disease and a marked improvement in the bronchiectasis.

N. G., aged sixteen years, also had a diagnosis of bronchiectasis. The maxillary sinuses were drained and the ethmoid sinuses were treated. The patient was discharged on February 7, 1927. When the patient returned, about a month later, she reported that she had slept well every night, while before the nasal treatment she hardly ever had a good night's rest. She had one attack of cough, which lasted four days and was not severe. Before treatment the cough was constant. The sputum is less than before. The weight has increased. The mother and daughter are delighted with the result. In this instance the result was secured without attacking the ethmoid labyrinth.

M. W., aged eight years, had a diagnosis of bronchiectasis. Before treatment of her paranasal sinus disease her temperature chart showed numerous spikes of temperature. Following treatment the fever disappeared. Dr. Jeans reported that the general condition of the patient was improved. This patient had her ethmoid sinuses operated upon. I regret to say that in the last week she returned to the clinic with pus coming from the ethmoid cells on one side of the nose. The bronchiectasis is doing fairly well. The maxillary sinuses, which had been operated upon several years before, were reoperated. Pus, polyps and necrotic bone were found.

The temperature curve of chronic paranasal sinus disease may simulate mild pulmonary tuberculosis. D. C., aged two years, suffered from loss of weight and his temperature ran from 98.8 to 102 degrees. After a careful study of the case the pediatrists convinced themselves that the tuberculous condition was healed and the clinical picture was due to an infection of the upper respiratory tract. With the treatment of the paranasal sinus disease the temperature curve became flattened and the weight increased. The reason for treating this child's sinuses was that the fever and loss of weight could not be explained on the basis of the chest condition.

Asthma in children is more benefited by treatment of the coexisting sinus infection than is the asthma in the adult.

G. O., aged nine years, had asthma for two years. The attacks were always preceded by an acute upper respiratory infection. They occurred in Spring, Fall and Winter; never in Summer. The faucial and pharyngeal tonsils were removed and the chronic suppurative paranasal sinusitis was treated, with no improvement. Intranasal drainage of the maxillary sinuses followed by treatment of the sinus condition was carried out. There was distinct improvement. The pediatrists were of the opinion that the sinus condition was responsible for the asthma. The ethmoid sinuses were operated upon. The Summer following this operation the mother reported that the child has had no asthma since the completion of the nasal treatment last Summer, although he has had one cold.

F. H., aged two years, had asthma for sixteen months. The attacks followed the taking of cold. All skin tests were negative. Nine months of treatment by the pediatrists gave no result. They were of the opinion that the sinus trouble which was diagnosed was the cause of the asthma. The faucial and pharyngeal tonsils were removed and the sinuses treated. The following Summer the mother wrote that the asthma was very mild and only present when she had a cold.

R. A., aged thirteen years, had a diagnosis of asthma. On August 17, 1921, he had a tonsillectomy and adenoidectomy. The asthma improved but persisted. On July 2, 1922, there was a diagnosis of hyperplastic ethmoiditis and sphenoiditis marked, and chronic maxillary sinusitis bilateral. A Sluder operation was done on each side. On August 22, 1922, he reported that he had one attack of asthma since the operation on the ethmoids and sphenoids. Each maxillary sinus was then operated upon. About a year later, July 17, 1923, he reported that he had, during the last twelve months, six attacks of asthma. On washing of the maxillary sinuses much pus was secured. The polyps in the ethmoid region were removed. The sinuses were treated and the polyps removed as they appeared until March 5, 1924, when the nose seemed to be quite clean. There has been no trace of asthma up to the present time. Cases of asthma in childhood should naturally be studied first by the pediatrist. If no cause for the asthma is found, the

laryngologist should be stimulated to make a very thorough examination of the paranasal sinuses. If sinus trouble is found it should be eradicated. One will use more radical treatment than he would use in an uncomplicated case. If, on attacking the sinuses, the asthma is improved but not eradicated, one should hold to the opinion until it is disproved that he has either overlooked a part of the sinus infection or he has not succeeded in eradicating it.

The presence of diabetes influences the treatment of sinus disease in several ways. In the first place, operations on the sinuses are much more likely to be followed by infection. Secondly, the patient should be well under the control of insulin at the time any operative work is done. Thirdly, if sinus disease is present the eradication of the infection is indicated just as much as the removal of diseased faucial and pharyngeal tonsils. Dr. Jeans has reported that as a result of treatment of chronic suppuration of the sinuses there has been a decrease in the insulin dosage necessary to control the diabetes.

B. D., aged five years, had a pediatric diagnosis of diabetes. The laryngologic diagnosis was chronic infection of the faucial and pharyngeal tonsils and chronic suppurative paranasal sinus disease. Following the removal of the tonsils the sinus disease was eradicated, and this resulted in the control of the diabetes by much smaller insulin dosage.

Last Summer we treated a number of infants suffering from diabetes twice a week under ethylen anesthesia for chronic suppuration of the sinuses, without the treatment or the anesthesia having the slightest deleterious effect so far as Dr. Jeans could determine.

If a child is a diphtheria carrier and the lymphoid tissue has been removed from the oro- and nasopharynx, that in itself is an indication for a careful examination of the paranasal sinuses and, if found diseased, their treatment.

K. O., two and one-half years of age, had been in the isolation hospital as a diphtheria carrier for several weeks. The faucial and pharyngeal tonsils were out clean before he became a diphtheria carrier. During the eighteen days that the patient was in the hospital as a carrier, the nose and throat were treated every day and the usual treatment for the eradication

of this condition was carried out. Then the maxillary sinuses were washed out under ethlylen anesthesia, and the next day the child was free of diphtheria organisms. On treatment of the maxillary sinuses considerable pus was found in the right antrum. The ethmoid region and the nose itself was found fairly clean. Certainly, if in children with a suppuration of the nasal sinuses the patients persist in remaining diphtheria carriers after the usual treatment has been done to eradicate the condition, the irrigation of the sinuses is indicated.

Recurring attacks of vomiting and fever and recurring attacks of fever without vomiting in children would indicate that the sinuses should be carefully studied, and if disease is found it should be eradicated.

I. H., aged seven years, came under treatment four years ago for recurring attacks of fever. He was referred by Dr. Byfield to determine the reason for the sudden elevation of temperature of 102° without apparent cause. The tonsils and adenoids had been removed one year before. The systemic condition was improved but the attacks of fever persisted. A chronic suppurative paranasal sinus disease was found. This was eradicated with permanent disappearance of the trouble.

F. M., aged six years, was also treated four years ago. He was referred by Dr. Byfield for the eradication of the cause of attacks of recurring fever with vomiting. A diagnosis of chronic suppurative ethmoiditis was made. This was eradicated four years ago, and since then he has had no recurrence of his trouble.

CONCLUSIONS.

1. The opinion of the pediatrist as to the probable presence of infection of the sinuses in the ears or both is most important.

2. The decision as to whether or not to attack the sinuses energetically, and if they are to be attacked, the time when it should be done is best made by the pediatrist.

3. No rules that are infallible for the treatment of paranasal sinus disease with systemic complications can be cited. Each case must be studied by the pediatrist and the laryngologist together, and the "clinical sense" of the two will decide what the treatment is to be.

4. The systemic conditions associated with paranasal sinus disease in infants and young children influence the choice of treatment. Each condition exerts its own influence.

5. They may indicate the need of immediate drainage of the maxillary sinuses, as in a very severe case with the cholera infantum syndrome.

6. They may indicate the necessity of avoiding traumatism of the mucous membrane, as in nephrosis and in diabetes except under insulin control.

7. They may indicate the need of radical procedures to remove the last vestige of paranasal sinus infection, as in arthritis, chorea and asthma.

8. They may indicate the need of several reexaminations of the sinuses in the more chronic systemic conditions and the treatment of the disease which is usually found on careful search.

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LXXXIV.

THE VARYING SYMPTOMATOLOGY OF CHRONIC MAXILLARY SINUSITIS, DEPENDING ON THE PATHOLOGY PRESENT.*

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The pathology of maxillary sinusitis has been worked out by Dmochowsky in adults and confirmed by Dean in children. On this basis it would seem as though the time had arrived when we might attempt a clinical classification of symptoms based on the pathology present at the time, not only for diagnostic purposes, but also as influencing our prognosis and treatment. This is of particular importance, as the writer believes that certain pathologic conditions in maxillary sinusitis are the basis of chronic invalidism or may cause death. Moreover, it should be possible to determine and remedy such conditions in time to save the patient. Such cases are not infrequent, and it is a practical question to not only make a diagnosis, but for rhinologists to come to some agreement on how such cases should be treated. For this purpose, let us analyze the accepted classification of chronic maxillary sinusitis to determine what cases give symptoms over which we need have no concern, and what cases require immediate and radical surgery.

CHRONIC MAXILLARY SINUSITIS AS THE RESULT OF A CATARRHAL PROCESS.

In chronic catarrhal maxillary sinusitis we have symptoms that are the result of an infection of low virulence, which involves the surface mucosa and is practically never associated with systemic complications. With such cases the important thing is to see that the patient is not left with a thickened

*Read at the meeting of the American Laryngological Association, Atlantic City, N. J., May 25, 1927.

antral membrane, which in most cases can be done by restoring the lymph and blood supply in the nares.

Pathology.—Zuckerkandl describes a catarrhal inflammation as one that is characterized by a serous infiltration of the mucosa whereby the membrane is increased in thickness some ten or fifteen times and appears jelly like. The exudate varies in amount and is mostly mucus. His opinion was formed on post-mortem findings. Uffenorde accepts this classification and applies it to all the sinuses. Hirsch questions whether there is a serous exudate or an infiltration, but otherwise accepts the definition.

Symptoms.—In chronic catarrhal maxillary sinusitis, the prominent symptom is a persistent unilateral or bilateral discharge. This may be diminished in amount, owing to infiltration of the mucosa about the ostium and to the thick mucoid character of the discharge. Pain and discomfort are rarely present. On account of the impaired drainage and the absence of symptoms directing attention to the antrum, chronic catarrhal maxillary sinusitis is usually overlooked or neglected until reinfection is followed by empyema.

Whether due to lack of aeration or to a low grade infection, there is increasing evidence that there are often many referred pains that result from such pathology, such as a general tightness of the head or through the eyes, pain over the eye or side of the head, inability to concentrate, etc. In addition, there is an increased susceptibility to colds and they are more persistent. As the pathology is confined to the mucosa, there is no history of systemic complications from acute exacerbations of a chronic catarrhal process.

CHRONIC MAXILLARY SINUSITIS AS THE RESULT OF A SUPPURATIVE PROCESS.

Clinically this form of chronic maxillary sinusitis can be divided into three classes:

First, those showing a thickened membrane and free pus.

Second, those showing a thickened membrane and no pus.

Third, those in which the lining membrane is undergoing a retrograde, degenerative process, and in which the secretion is serous or gelatinous.

CHRONIC MAXILLARY SINUSITIS WITH A THICKENED LINING MEMBRANE AND PUS (EMPYEMA).

Before an empyema occurs there has usually been a gradual thickening of the nasal mucosa, as the result of an hypertrophic process or a previous suppuration secondary to some dental cause, which has impaired drainage to such a degree that a virulent process has resulted in a partial or complete closing of the natural opening.

Symptoms.—With a thickened membrane and pus present, in the chronic form of maxillary sinusitis, there may be but few symptoms beyond the purulent nasal discharge and the pharyngeal irritation. With any exacerbation of the chronic process there is discomfort over the affected antrum, or even pain, with an increase in the unilateral discharge, although marked pain is rare. The affected side of the head may ache or supraorbital neuralgia may be present. In the afternoon there may or may not be an increase in temperature of one or two degrees. The character of the discharge varies from a fetid secretion, due usually to dental necrosis, to a mucopurulent or purulent type. If there has been a purulent discharge over some time, there will be dryness of the mucous membrane over which it passes. Such patients will often complain most of their throats, and the difficulty in keeping them free from thick secretion or crusts. Often there is a persistent cough on lying down and marked pharyngitis. The sense of smell is not often disturbed, unless there is an associated ethmoiditis or marked swelling of the middle turbinal that shuts off the upper vestibule of the nose. In many cases the progress of the disease may be characterized by a unilateral discharge and marked myostitis affecting different muscle groups. As the pathology is found largely in the mucosa and connective tissue, secondary involvement of distant organs is not so common, although acute exacerbations may be followed by disastrous results, especially in organs the seat of some chronic inflammatory process. Again, when empyema is present, this does not preclude a previous chronic inflammation and blood stream infection that makes the opening of such an antrum dangerous as distinguished from the opening of an acute empyema when the mucoperiosteum is not involved.

Antra With a Thickened Membrane.—Rev. H. A. A., B. Iowa, 40 years, American, married. Referred by Dr. S. on account of continued headaches, April 13, 1925. P. H.: Measles, pneumonia, appendicitis and jaundice. Patient also had had two or three attacks of what he called pleurisy, lasting several days, with a previous infection of his right antrum. At this time he gave a history of a cold for three months, with pain in the head and right nasal discharge on leaning forward for one month. Head pain interfered with mental work. Catarrh until he went to college but not since. There was a history of tonsillitis as a child, with a tonsil and adenoid operation in 1920. Thick nasal discharge in the morning.

Examination: Septum to extreme right. Right antrum dark on transillumination. Temperature 99.8°, pulse 84, respiration 24.

Diagnosis: Right chronic antrum with an acute exacerbation.

Treatment: Right antrum was opened under gas oxygen anesthesia and a wide opening made in the antranasal wall and sinus irrigated with warm saline. That night patient was chilly and had a temperature of 103° with considerable pain in his head and thick discharge. The following day the temperature continued, and irrigation was followed by thick purulent discharge. Patient now commenced to complain of cramps in his right leg and extreme pain on moving it. The third day the leg pain became worse, and a surgeon made a diagnosis of an embolus and said amputation would be necessary.

That night the patient died suddenly.

Note.—Although the patient gave no history of an acute exacerbation for three months, yet there was probably an active infection of the deeper tissues present at the time of the operation. A diagnosis of empyema would have been made without the history.

CHRONIC MAXILLARY SINUSITIS WITH A THICKENED LINING MEMBRANE AND NO PUS.

Symptoms.—When a thickened membrane is present in the maxillary sinus without the presence of pus, the symptoms are due to acute exacerbations which simulate nasopharyngitis but are apt to be more prolonged. Sometimes there is an uncomfortable feeling on the affected side, but the rule is

that all subjective symptoms are absent. In the early stages there will be a unilateral mucoid discharge, and the nasal mucosa will be injected on the affected side. In some cases there is a history that several years ago there was a purulent discharge which of late has been watery. There may be no temperature or it may be elevated one or two degrees. There may or may not be tenderness over the canine fossa on pressure. In the same way, pain over the eye or side of the head may or may not be a prominent symptom. There is usually sensitiveness to palpation of the gland under the angle of the jaw. The roentgenogram shows a thickened membrane. On account of the absence of subjective symptoms and of free pus in the antral cavity, its relation to systemic complications is often overlooked. It is in these cases that a careful history, told in the patient's own way, will show that over a long term of years his socalled colds have been followed by bronchitis, myositis, arthritis, etc. This is important, for a blood stream infection having already occurred, acute exacerbations of the chronic process in the antrum have been followed by myositis, erysipelas, arthritis, endocarditis, glomerulonephritis, serous labyrinthitis, involvement of the gall bladder, appendicitis, etc. These complications are more common in this form of sinusitis than when an empyema is present. If, following an acute exacerbation, there is a difference in pressure over the canine fossa some three or four weeks later, it is probable that a rarefying osteitis is present in the antral wall, and such cases are almost sure to have serious complications whenever the patient's resistance is lowered, if such a history has preceded it. While a thickened membrane may exist for years in the antrum without symptoms, except those of repeated colds, if there is a history showing systemic infection, such pathology in the deeper structures of the wall of the antrum may be followed by complications that eventually may lead to chronic invalidism or death. These cases are not uncommon, and the symptomatology outside of the referred pains is almost wholly that of the systemic complications that have resulted from the blood stream infection following acute exacerbations of the local process.

Pathology.—When thickened membrane is present in the antrum and no pus, the pathologic examination, where sys-

temic symptoms have followed acute exacerbations of the sinusitis, has shown chronic inflammatory reaction of the mucosa and periosteum, with a noticeable increase in the fibrous tissue. Invasion of the bone and thickening of vessels entering the bone may occur.

Antra With Thickened Membrane. Case 1.—Mrs. T. J. C., 52 years, housewife, American, seven children. Referred by Dr. T., September 15, 1926, for cause of recurring eruption on her chin.

P. H.: Rheumatism, appendicitis, tenderness over the gall bladder, skin eruption, erysipelas. Has had frequent sore throats for thirty years. An eruption on her chin, 2 by 4 cm., has been rebellious to treatment, and after healing would recur following her sore throat. Nine years ago had a severe attack of rheumatism and her knees were put in splints. Has been lame at times since. Fatigue marked. Following her last sore throat she was sensitive over the gall bladder. One year ago erysipelas commencing on the left side spread over the entire head with a temperature of 105°. Has considered herself an invalid for five years.

Examination: Transillumination of the sinuses shows the left antrum dark with eye reflex absent. Pus in the nares on the same side and mucosa injected. Palpable glands on the left side. Antrum sensitive to pressure (no recent exacerbation).

X-ray: Increased density of the left maxillary sinus and a highly calcified area on the left side of the frontal sinus. Area of infection on the left lower incisor.

Clinical diagnosis: Left maxillary sinusitis with a thickened membrane and osteitis. Chronic tonsillitis.

Operation, September 29, 1926, Brooks Hospital: Atropin, grs. 1/150, gas-ether anesthesia. Postnasal packing inserted. Usual incision and entrance to the left antrum through the canine fossa. Mucous membrane lining was found thickened. Nasoantral wall taken down. Lining membrane removed. Cavity packed with vaselin gauze. Incision closed by three catgut sutures. Postnasal packing removed. Tonsillectomy performed. Closed abscess found in the right tonsil. One suture required in each fossa. Pressure bandage applied.

Postoperative: Had pain in her face which was marked, with decreasing intensity for three weeks following the operation. November 30, 1926. Dismissed free from symptoms.

Note.—May 6, 1927. Has had a good winter and is in better physical condition than for a long time.

Antra With Thickened Membrane. Case 2.—Mrs. A. A. D., 58 years, housewife, American. May 10, 1925. Referred by Dr. R. on account of severe headaches and continuous afternoon temperature.

P. H.: Membranous croup (?) at 12 years. Bronchitis each fall. Mastoiditis. Never strong; 15 years ago had grippe three times in successive years and several attacks in later years; diphtheria at 12 years; 10 years ago patient had an attack of shingles and bleeding gums, and from this time she dates her trouble. She has had chills at frequent intervals for ten years, without acute infections accompanied by pressure in the head. Vertigo. Burning pain over right eye. Throbbing pain back of the nose which comes on after being up for an hour or two, then the nares would fill, followed by purulent discharge. The discharge of late has been thinner and more mucoid. There would be an afternoon temperature of 100° for weeks, loss of appetite, insomnia and marked nervousness until the discharge took place. Has lost 40 pounds. Two years ago there was a left radical antrum and exenteration of the ethmoid cells with no discharge since, but a constant recurrence of chills, temperature, headache, nervousness, loss of appetite and insomnia. Abscessed tooth removed one and one-half and two years ago.

Examination: Patient consulted the writer May 10, 1925. She is poorly nourished, nervous and apprehensive and looks ten years older than her age. Left ethmoid cells and antrum have been exenterated. Frontal sinuses negative. Right ethmoid cells exenterated and sphenoid opened. Right antrum tender on pressure. Transillumination negative. No free secretion in the nares or postnasally. Physical examination negative.

X-ray: Right antrum contains thickened membrane.

Clinical diagnosis: Right chronic maxillary sinusitis. Chronic tonsillitis.

Operation, October 20, 1926, Massachusetts Women's Hospital: Atropin, grs. 1/150. Gas-ether anesthesia. Postnasal packing inserted. Double radical antrum operation. Incision made in the usual manner through the canine fossa. Mucous membrane of both antra was found slightly thickened and stripped readily from the bone. The left antrum showed the nasoantral wall opened by a previous operation. Vaselin gauze packing. Incision closed with two catgut sutures. Postnasal packing removed. Tonsillectomy. One suture required in the right fossa. Pressure bandage applied.

Note.—Premature senility is a bad surgical risk. This patient had a cardiac attack on the third day following operation. Since then she has steadily gained and been free from chills, temperature and headache. As cold weather came on an acute infection involved the antrum with a temporary return of her symptoms. This has happened several times, as is usual in these chronic cases for the first year. Between attacks she is free from symptoms and has gained seven pounds.

Antra With Thickened Lining Membrane and Beginning Degenerative Changes. Case 3.—Mrs. J. B. A., age 54, housewife, American. Referred by Dr. M., June 24, 1925, for nasal obstruction.

P. H.: Bronchitis, asthma, grippe. Scarlet fever 23 years ago. Grippe several times. Colds and bronchitis, but not as often as formerly. Fatigue marked. Asthma for twenty years.

Examination: Septum to right. Left mucosa injected. Left middle turbinate has been partially removed. Transillumination dark over the left antrum and eye reflex not present. More discharge from the left nose. More tender over the left canine fossa. No adenopathy. Tonsils show chronic infection.

X-ray: All sinuses clear except the antra, in both of which there is a very slight thickening of the membrane. On the left there is sclerosis of the bone under the lining, suggestive of some previous infection.

Clinical diagnosis: Right chronic antrum with degenerative lining membrane. Left chronic antrum with thickened lining membrane and osteitis.

Operation, June 30, 1925, Brooks Hospital: Atropin, grs. 1/150. Gas-ether anesthesia. Double radical antrum operation.

Polypoid membrane found in both antra. Vaselin gauze packing and incision sewed with catgut sutures. Tonsillectomy. Two sutures required in the left fossa.

Progress: April 15, 1927. Twenty-two months after operation the patient states that she has had but one attack of asthma and has had a better winter than for years. No antral discharge.

Antra With Thickened Membrane. Case 4.—Mrs. H. S. D., 32 years, B. Maine, housewife, American. History of neuritis, myositis, appendicitis and frequent colds. Referred by Dr. C., May 13, 1926, for neuritis in arms and shoulders and frequent colds.

P. H.: Grippe, rheumatism three months, myositis several times. Measles, mumps, whooping cough as a child. Later she had typhoid fever. There was a history of grippe and rheumatism for three months followed by lameness and pain in her arms and shoulder. Throat is rough without hoarseness, and acute exacerbations have been accompanied by otalgia. Thick discharge in morning. Breath bad. Fatigue marked. Does not hear as well in the right ear. Singing tinnitus located in the head. Lost 12 pounds. Operated upon 12 years ago and thinks she is a bleeder. Ectopic pregnancy operation and appendectomy.

Examination: Septum to the right, adenopathy, especially on the left side, with tenderness to pressure. Right maxillary sinus transilluminates poorly and is sensitive to pressure. No postnasal discharge. Third tonsil enlarged, especially on the left side. Both membrana tympani indrawn. Light reflex broken with thickening of the posterior fold and over Shrapnell's membrane.

X-ray: Right antrum more dense. Teeth normal.

Diagnosis: Chronic tonsillitis. Right chronic antrum with osteitis (?).

Operation, May 15, 1926, Brooks Hospital: Atropin, grs. 1/150. Gas-ether anesthesia. Postnasal packing inserted. Right radical antrum operation. Vaselin gauze packing. Incision closed with two catgut sutures. Tonsillectomy. One suture in the right fossa, two in the left. Postnasal packing removed. Pressure bandage applied. Patient poor breather through entire operation. Pulse remained normal.

Progress: One year after operation patient reports a gain in weight of 22 pounds. She is free from general symptoms. She has had no antral discharge until a recent infection. Was married seven years, and on account of poor health she had decided that there was no prospect of having a family and adopted a child. Patient expects to be confined in June.

Case 5.—Mr. J. L. A., 38 years, B. Massachusetts, engineer, married. Referred by Dr. S. on account of vertigo and deafness, June 16, 1922.

P. H.: Quinsy sore throat. Bronchitis. Labyrinthitis. Neuritis eighth nerve. Measles, mumps, diphtheria, scarlet fever, bronchitis, quinsy sore throats. Tonsil and adenoid operation eight years ago. Had been working until late at night for some time when he was taken with an acute infection on April 6th, accompanied by pain over the right eye, temple and in front of the ear. The hearing was impaired slightly for a few days. There was nausea and vomiting. The canal and auricle were swollen, with a blister at the entrance to the canal. In bed four days. On getting up patient was dizzy. Could not walk without support and since then there has been a marked tinnitus. No previous aural history. Saw a prominent aurist May 10th, who did the Bárány tests and made a diagnosis of eighth nerve deafness. No previous history of stomach trouble. June 16th, over two months later, Mr. A. consulted the writer, complaining of still being unstable and of pressure in the ears, with a tendency to fall to one side. Bárány test negative. Ears feel stopped up. Tinnitus like steam. Vertigo slight and not influenced by position of head. No nystagmus.

Examination: Both membrana tympani indrawn and in contact with the promontory. High deviation of septum to right. Previous fracture between quadrangular cartilage and the maxillary ridge. Transillumination too clear over the right antrum.

Clinical diagnosis: Eighth nerve deafness secondary to an acute exacerbation of the right chronic antrum with secondary degeneration of the lining membrane. Serous labyrinthitis and pemphigus two months ago.

X-ray: Increased density over the maxillary sinuses, especially the right, and the right ethmoid. Frontals normal. Teeth normal.

Operation: Right Caldwell-Luc at Massachusetts Eye and Ear Infirmary. Ether anesthesia.

Examination, September 25, 1923: Free from symptoms and hearing normal.

September 25, 1923.			October 17, 1922.		
R.		L.	R.		L.
25/25	W. V.	25/25	9/25	W. V.	25/25
55/14	Rinne	58/17	16/6	Rinne 256 C^1	21/8
+	W.		—	W.	—
N	U. L.	N	N	U. L.	N
32	L. L.	32	32	L. L.	32

Antra with Thickened Lining Membrane. Case 6.—Mrs. M. Y., 47 years, housewife, American. Referred by Dr. G., July 8, 1926, for injection of nerve on account of pain over the eye, back of the ear and over the right antrum. Duration, seven years.

P. H.: Trifacial neuralgia from five to seven years. Trifacial pain, intermittent in character, which is present constantly as an ache, but is described as "shooting," with exacerbations which may be brought on by wind, change in temperature or acute infections. Three branches of the nerve were involved, which commenced as a pain over the right eye. Right unilateral discharge. No bad taste or odor. Sense of smell lost. Right upper lip sensitive. Discharge thick and yellowish. Right ear sensitive and hearing diminished. Sensitive to light and deep pressure over the canine fossa on the right side. Insomnia, but has not used hypnotics, as morphin makes her sick. Lost flesh at first, but has gained some weight during the last year. All the upper teeth have been removed. Has had several operations through the nose and one through the canine fossa. The pain was better for a time while the antrum was discharging. Has been an invalid for the last five years.

Examination: Patient poorly nourished, nervous and apprehensive. Transillumination dark over the right antrum anteriorly but clear through the base. Frontals clear. No anterior or posterior nasal discharge. Sensitive to light and deep pressure over the right antrum. Pain follows the distribution

of the trifacial nerve. Right upper lip sensitive and also the right ear. Right nasal mucosa injected. There is an opening through the antronasal wall the size of a one cent piece.

X-ray: On account of having had several operations on the antrum, roentgenograms were not taken.

Diagnosis: Right chronic antrum with thickened membrane probably not wholly removed.

Operation, August 11, 1926: The operative field was explored through the right canine fossa. The antral cavity looked clear and free from unhealthy granulations. There was an opening anteriorly through the antronasal wall the size of the little finger, round, with cleancut edges. On examining the anterior and superior sinus wall, a thick membrane was found over one-third of its surface. This was carefully removed and the antronasal opening extended back to the junction of the posterior antral wall. A vaselin strip packing was introduced and the end carried into the nose. The cheek incision was closed and a compression bandage applied. A shock enema was given.

Progress: August 30, 1926: Patient has slept all night since the operation, is eating well and is free from pain.

Note.—This case had free drainage and aeration, but her symptoms continued until the diseased lining membrane was removed. Although her trifacial neuralgia had existed for seven years, she had no pain following the operation for five weeks. Two weeks after her return home she had an acute infection with return of the pain. Since then she is free from pain for a week following each treatment. This patient should have been under observation for three or four months.

Case 7.—Mrs. F. S. D., 52 years, B. Maine, housewife, American. Referred by Dr. D., who found evidence of some focal infection in the eyes.

P. H.: Rheumatism three months. Mumps and chickenpox as a child. As a young girl she had postnasal discharge, which was quite thick, but of late years the discharge has been anterior and watery, especially on the left side. Has a tendency to clear the throat. History of tonsillitis. Since adult life she has had myositis at different times, and three years ago the knees were swollen, requiring bandaging, and at this time there was soreness over the gall bladder. Noticed spots before the

eyes six years ago on the left side and now for two weeks in the right eye.

Examination: Patient well preserved, of good color, and not old beyond her years. Weight normal. Septum deviated to right. Mucosa injected on both sides and slight amount of mucus on both inferior turbinates. Transillumination more clear on the left side. Right tonsil shows cryptic disease but no pus. Glands palpable on both sides. Pharynx clear.

Clinical diagnosis: Bilateral chronic maxillary sinusitis with thickened membrane on the right side and beginning atrophic changes on the left side, with no clinical evidence of osteitis.

X-ray: The frontal sinuses appear normal. The ethmoid cells appear clear. The right maxillary sinus appears denser than the left. This is more marked in the A. P. view than in Water's position. When only examining the picture from Water's position, and not knowing history, one might almost say the appearance is within normal limits.

Operation, November 23, 1926, Massachusetts Women's Hospital: Atropin, grs. 1/150. Gas-ether anesthesia. Postnasal packing inserted. Right antrum opened through the canine fossa. Mucous membrane stripped readily from the bone. Nasoantral wall removed, antrum packed with vaselin gauze. Incision closed with two catgut sutures. Left antrum opened also. Mucous membrane found thin and atrophic. Vaselin gauze packing. Two catgut sutures. Postnasal packing removed. Pressure bandage applied.

Note.—When the thickened membrane has undergone atrophy so that it is about normal in thickness, the roentgenogram is of but little value. The history with the local findings were sufficient for a diagnosis.

CHRONIC MAXILLARY SINUSITIS WITH POLYPOID DEGENERATION. PATHOLOGY.

On inspection after an opening through the canine fossa the mucoperiosteal lining may look smooth, may be covered with small, multiple abscesses, a polypoid mass be found in the floor of the antrum or opposite the middle turbinal body, the cavity may be filled with serum or contain a gelatinous

mass. The whole mucosa is undergoing degenerative changes, is thin, and shows chronic infection. Pus may be found between the mucoperiosteum and the bony wall or an osteitis be present. There may be a thickening of the wall of the blood vessels entering the bone. Sometimes the whole cavity is filled with mucous cysts. While most cysts contain clear serum, occasionally one will contain mucopus tinged with blood. Polypi may be of the mucoid type or contain a large amount of fibrous tissue, or become calcified.

Diagnosis: If we see the patient during an acute exacerbation, there will be evidence of a low grade infection of the nasal mucosa, on the affected side, with beginning atrophy. The discharge is unilateral and serous. The ostium is larger than normal, from atrophy of the surrounding soft tissues. There is usually a lateral pharyngitis. During the acute phase, but not at any other time, there is soreness on pressure over the canine fossa, although there are exceptions to this rule, and the gland under the angle of the jaw is sensitive to pressure and may show hyperplasia. On washing out the antrum, a gelatinous mass is often found, or only a serous fluid is present, or the lavage may be negative. Transillumination of the diseased antrum is more clear than normal on the affected side. The roentgenogram report is usually a thickened membrane on the healthy side, and is of value for its negative evidence. If polypi are present, these may show in the picture.

Symptoms: These patients are usually free from symptoms until they have an acute exacerbation, which they consider a cold, and to which they are susceptible. The discharge, being serous, is not noticed, and the symptoms are those of a subacute nasopharyngitis. The recurrence of these symptoms may continue for years, or, in exceptional cases, the acute phase may be accompanied by albumin in the urine and symptoms of general toxemia, as in the cases with a thickened membrane. Usually there is no pain over the affected antrum. It is in these cases, where the thickened membrane is undergoing degenerative changes, that we so often have vasomotor symptoms with the exacerbations, but between times they consider themselves free from any infection.

Treatment: In empyema it is advisable to use every means at our command to cure our patient with the least damage pos-

sible to the tissues, in the hope that function may be restored without resorting to a radical operation. In the types where there is thickened membrane, and where this has undergone degenerative changes, the mucoperiosteum is involved, and when we have systemic symptoms following acute exacerbations of the local chronic inflammatory process, the entire removal of the lining membrane is the only treatment. This should be an interval operation, not earlier than four weeks after an acute phase. On account of the involvement of the deep tissues, it is evident that simple ventilation and drainage must be inadequate after a blood stream infection has occurred.

Antra Showing Chronic Inflammatory Changes in the Lining Membrane, With Secondary Atrophy.—Miss E. M. M., American, nurse, age 27.

Complaint: Constant colds, specks before the eyes, with repeated attacks of myositis.

P. H.: Grippe, bronchitis, pneumonia, appendicitis, rheumatism, measles, mumps, whooping cough, chickenpox, bronchitis, pneumonia, rheumatism, appendicitis and grippe. Always had colds and bronchitis nearly every winter. Occipital headaches as a child. Laryngitis since 15 years of age. Tonsil and adenoid operation in 1923. Has had no loss of voice since but gets very hoarse. Bronchitis in 1923, with an attack of acute appendix. No operation. In 1920, lobar pneumonia, and in 1921 bronchopneumonia. Acute antrum in 1921 and 1924 on the left side. Myositis in back and shoulders and a good deal of foot trouble (flat feet). During the last two years has had constant colds and hoarseness, especially when tired, with repeated attacks of myositis. Has been having specks before her eyes, which the ophthalmologist says is due to infection. Headaches four or five times a week.

Examination: Left nasal mucosa injected. Transillumination too clear on the left side and through the base. No tenderness on pressure. Palpable gland in the neck. No free secretion in the nasopharynx. Right nares negative.

X-ray: The frontal sinuses are small. The other sinuses are perfectly clear. (Macmillan.)

Blood: Hemoglobin 87 per cent, red count 4,080,000, white count 8,355. Differential: Polymorphonuclears 64 per cent, large lymph. 28, small lymph. 7, mononuclears 5, mast 5.

Clinical diagnosis: Left chronic maxillary sinusitis with beginning secondary atrophy.

Operation: Radical Caldwell-Luc operation under ether anesthesia, and entire lining membrane removed. Upon removing bony wall the membrane covering the face of the antrum stripped from the bone, ballooning into the antrum. Bone and membrane sent to the laboratory.

Laboratory report: Specimen submitted shows chronic inflammation of the mucosa and periosteum, with some inflammatory reaction in the blood vessels of the bone. There is no reaction in the bone tissue proper. (Slack.)

Double Chronic Maxillary Sinusitis With Secondary Atrophy on the Right Side.—Miss E. B., 38 years, teacher, American. First seen January 3, 1927, with question of some focus of infection.

P. H.: Tonsillitis followed by repeated attacks of myositis. No colds until last winter. Influenza 1919 (mild case). In 1923 commenced to have trouble in voiding. No sore throat preceded it. Bad taste. Six years ago had indigestion and has to be careful now about her diet. Occasional acidity. Has had myositis in her right shoulder and is having a little stiffness of the neck now. Has severe generalized headaches, accompanied by pain in the teeth on the right side. Tongue gets coated. Has a bad taste and stomach is upset as often as once a month for the past fifteen years. Face is sore with her colds. Backache every night. Examined by two rhinologists, who pronounced the nasopharynx and sinuses negative.

Report of general examination: Two years ago complained of frequency of urination and bladder pain. Examination locally showed ulcer in the apex of the bladder. Local treatment for a year was followed by no improvement. June 14, 1926, a large portion of the apex of the bladder containing two ulcers was resected. There was some improvement in the pain and frequency of voiding, but the urine continued to show infection and the results were only partially satisfactory. Examination November 20, 1926, showed definite strictures in the lower end of both ureters and pus coming from both kidneys. The ulcers had healed and the bladder showed no other trouble. During these sixteen months she had been referred to two

rhinologists and a dentist, who failed to find any source of infection, although the urologist felt that this class of case always results from some primary focus.

Local examination: Left neck showed palpable tonsillar gland. Both tonsils show chronic infection. Right antrum is more clear to transillumination, especially through the base. Nasal mucosa negative. No pain on pressure over the canine fossæ. Right postnasal spur. No postnasal secretion and none in the nares.

X-ray report: Left mucoperiosteum definitely thickened in the maxillary sinus. (Macmillan.)

Urine examination, January 7, 1927: Reaction slightly acid. Specific gravity 1006. Sugar none. Acetone none. Albumin slight trace (Heat, HNO_3). Sediment small amount. Blood rare changed corpuscle. Pus small amount. Crystals none. Casts none. Many squamous cells. Rare large round epithelial cell.

Diagnosis: From the clinical history, confirmed by the finding of a palpable tonsillar gland on the left side, and local evidence of chronic infection, together with evidence of abnormal clearness to light through the right antrum where she had the pain in her teeth, and the evidence from the X-ray of a thickened membrane, a diagnosis was made of chronic tonsillitis and double chronic maxillary sinusitis, with probable beginning secondary atrophy on the right side.

Operation, January 7, 1927: Tonsillectomy and double radical antrum through the canine fossa, under general anesthesia. Entire lining membrane was removed and this, with a section of bone, was sent to the laboratory. Mucoperiosteum thicker on the left side, and both sides show chronic infection of the deep tissues without involving the bone. (Slide) (Slack).

Postoperative result: Feb. 17, 1927. Urine examination the same, except an increase in specific gravity to 1010 on three examinations. Backache relieved at once and patient has had but one headache. Pain on voiding gone but frequency remains the same.

SUMMARY.

Chronic catarrhal maxillary sinusitis is of clinical importance only in the fact that it leaves a thickened membrane that im-

pairs drainage, and if a hypertrophic process is present in the nasopharynx the conditions are right for a more virulent infection to be followed by an empyema.

Chronic suppurative maxillary sinusitis involves the tissues superficially, and the danger to remote organs in most cases is not marked as long as the pathology is confined to the lining membrane; but if the periosteum is involved, then the danger of a blood stream infection obtains, as is present in the two following subdivisions:

With a thickened membrane, or one undergoing a secondary atrophy, any change in the circulation of the blood and lymph at the base of the skull would naturally be followed by reflex symptoms. Stasis, pressure or any condition which would cause hyperemia often are the cause of vasomotor symptoms, a feeling of tightness about the head, bad feeling head, and often an indefinite feeling that patients find hard to describe, but which makes concentration difficult. Actual pain is usually supra-orbital, but may be through or below the eye, or simulate a trifacial neuralgia. Changes in temperature and fatigue induce or aggravate their symptoms.

Chronic maxillary sinusitis with a thickened membrane, or a membrane undergoing degenerative changes, was for many years supposed to be of but little clinical significance. There is increasing evidence that on account of the pathology involving the mucoperiosteum, these cases are a menace to the health or even the life of the patient. No examination, however thorough it may be, is of any value unless it is checked up with a detailed general history told in the patient's own way. Any surgery done during the acute phase, when there has been systemic complications through the lymph channels, may result in death to the patient. Following years of chronic infection, there are changes in the blood cells and usually a myocarditis present. Such patients look old beyond their years, and a prolonged operation may be followed by heart failure.

Among the chronic antra operated upon by the writer during the last seven years, in twenty cases there were systemic symptoms severe enough to cause invalidism. Conservatism should always be observed in any surgery of the nasopharynx or sinuses as long as there is a possibility of restoring function.

In these chronic cases of sinusitis, where the mucoperiosteum is involved, which has been followed by systemic complications, the only conservatism is entire removal of the lining membrane.

Examination of the mucoperiosteum shows in all these cases chronic inflammation, and in some cases the blood vessels that run into the bony wall are enlarged. Section of the bony wall and lining membrane confirm the pathology so well given by Carmody in his paper of 1925 and previously given by Dmochowsky.

LXXXV.

THE RELATION OF PREGNANCY TO MASTOID INFECTIONS.*

By Harold A. Fletcher, M. D.,

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One of my patients had an acute exacerbation of an old apparently healed mastoiditis in the first few weeks of her second pregnancy. The mastoid symptoms immediately became alarming, with fistula formation, and remained so until a spontaneous abortion took place, a week after radical mastoid operation. The wound healed in a reasonable time until, in the first two weeks of her next pregnancy, there was again an acute exacerbation with alarming symptoms. She was reoperated upon, but the mastoid did not heal until after the third operation, together with termination of labor by Caesarean section eight months later. During this pregnancy her mastoid and head symptoms became increasingly worse until the time of her Caesarean operation, after which mastoid and head symptoms again entirely disappeared and the mastoid wound healed, with the exception of one small area. Seven months later, in the first weeks of her next pregnancy (third), the ear again flared up with alarming symptoms, which cleared up immediately on termination of the pregnancy, and the mastoid wound healed and has been healed for six months. I reported the case briefly at the California State meeting in 1925. Since then this patient has had one short period of pregnancy, terminated after two months by a self induced abortion followed by a severe pelvic inflammatory process, which later necessitated a total hysterectomy. During this last pregnancy the ear symptoms were beginning to arise, but the mastoid had not broken down when abortion was brought on.

Previous to my experience with this patient, I had not heard that pregnancy had any particular effect on ear infections nor

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had I read of any reported cases. In looking carefully through the literature, to my surprise I have found that as far back as 1879 pregnancy was occasionally suspected of causing or held responsible for flareups in ear infections, yet the numbers of authors to date is only fifteen.

J. Baratoux,[1] a Frenchman, in 1879; K. Lichtenberg,[2] in Budapest, in 1880; Bürckner,[3] in Germany, in 1880, and Weber-Liel,[4] in Berlin, in 1883, were the first to mention the relationship. Apparently separately and unknown to each other, the first two almost simultaneously published papers on the influence of various pathologic and physiologic conditions of the female generative organs on the ear, among which pregnancy and middle ear infections are mentioned.

From this time until 1910 various men wrote on the subject. Thomas McArdle,[5] 1888, reviewed Baratoux and Lichtenberg's work and mentioned cases of his own. Haugh,[6] 1893, mentions it in his textbook on the ear. Fellner,[7] 1903, published a case in point. Seitz,[8] 1907, mentions the relationship in Döderlein's textbook on obstetrics, and Mauvoisin,[9] 1907, mentions it briefly. In all of the articles referred to above, the cases reported were very indefinitely given, with vague clinical findings, and are of interest only in that they give rise to a suspicion in the minds of the writers that pregnancy was the cause of the ear complications, although in no way proving such to be the case. Fellner's case, 1903, was the only one giving any real clinical data to judge by. This patient developed an acute otitis media purulenta with mastoiditis two days before term, followed by lateral sinus thrombosis, and died three days later with septicemia.

In 1910, Heermann[10] published five cases. His group of cases was the first which really gave detailed histories and clinical findings and attempted to show the unfavorable effect of pregnancy on middle ear infections. His first patient had a chronic mastoid with temporal lobe abscess, both of which cleared up on operation before normal termination of pregnancy. This patient was also reported by Heine.[11] The next three patients had chronic middle ear infections with mastoiditis, which responded to radical mastoid operation during pregnancy and were healed before term. His fifth patient had an acute otitis media and acute mastoiditis during second

pregnancy, necessitating simple mastoid operation, which healed during pregnancy but healed abnormally slowly. The same patient in her next pregnancy had an acute otitis media in the operated ear, which did not require operation, as the previous operation had been a modified radical mastoid operation preserving the drum but also leaving a permanent opening above, posterior and lateral to the drum. This otitis media did not heal until after labor. In the fourth pregnancy another acute flareup occurred, and Heermann asks if abortion is not indicated in these cases, as in tubercular laryngitis and eclampsia. He is the first to ask the question.

The next paper published on this question was one by Bondy and Neumann[12] of Vienna, in 1919. They mention Heermann's five cases and publish four of their own. The first was a patient with a bilateral acute otitis media in her fifth month of pregnancy. The ear condition went rapidly to formation of fistula in the right ear. This case had in addition an active tuberculosis of the right upper lobe with some redness of the arytenoids and paresis of the transversus and internus muscles of the larynx. Abortion was performed. Ten days after abortion the left ear was almost dry and there was no vertigo or fistula symptom. When this patient was next seen, six months later, the left ear was dry, but the right ear had been discharging for the past two weeks. They believe that the abortion definitely helped the ear condition.

The second case reported was a woman, four months pregnant, who developed an acute otitis media, which progressed rapidly to an acute mastoiditis with vertigo and marked tinnitus. A simple mastoid operation was done, which caused no cessation of the foul discharge. Two and a half months later facial paralysis occurred, and three and a half months after the simple mastoid operation a radical operation was performed which did not heal, areas of bony necrosis reappearing, until delivery. Three weeks after delivery the wound healed, except for a slight granulation tissue in the region of the semicircular canal.

The third patient was a woman in her fourth month of pregnancy, who had had an acute exacerbation of a chronic otitis media when two months pregnant, with the gradual develop-

ment of headaches, dizziness and a fistula symptom in the left ear. Abortion was advised but she left the clinic and was lost from observation without anything being done. She had, however, no history of acute exacerbation before this pregnancy, and they place responsibility of this on pregnancy.

The fourth patient reported increased suppuration of a chronic otitis media left ear in her first and second pregnancies, which subsided after delivery each time. During the eighth month of her third pregnancy increased discharge and pain occurred in the left ear. A radical mastoid operation was done eighteen days later, because of increasing pain and headaches. Dura and sinus were found exposed at operation. Cholesteatoma present. She developed postoperative lateral sinus thrombosis eight days later, which was opened, drained and ligated. Thirty-four days after lateral sinus operation, marked dizziness occurred when wound was dressed and positive fistula symptom was present. One week later labor was induced, with prompt cessation of mastoid and labyrinth symptoms and complete healing of mastoid wound, two months later.

Bondy and Neumann then conclude that pregnancy can have a definitely unfavorable influence on existing suppuration of the middle ear in both early and late months of pregnancy. They also claim that abortion is to be considered if during pregnancy the ear symptoms threaten complications, and go so far as to say that these cases would not respond to radical operation without abortion.

The next three cases recorded are by Fremel,[13] also of the Neumann Clinic, Vienna, in 1920 and 1921. His first two cases are unimportant ones, having an acute purulent otitis media with complications, both being in pregnant women but showing no relation of the process as regards pregnancy and no report being given of the effect of labor or termination of the pregnancy on the process.

Fremel's third patient, however, was a case in point. The patient was seven months pregnant and had an acute purulent otitis media of five weeks' duration. At operation there was found extensive destruction of bone, exposing the sinus and the dura of the middle fossa. A fistula symptom developed

fifteen days after operation, and the mastoid wound showed no tendency to heal. After induced labor, at which time the radical mastoid operation was done, the fistula symptom cleared up and the wound healed in six weeks.

The next to write on the subject was Blohmke,[14] 1922. Blohmke writes a very extensive article, reviews all of the previous cases mentioned and publishes one of his own. The patient was treated for a purulent otitis; then a radical operation under local anesthetic was done, at which localized meningitis was found. The emissary vein was torn at operation and was tamponed. Sinus was negative. Normal labor came on following operation. The patient developed puerperal infection and died of peritonitis secondary to para- and endometritis.

Blohmke then takes up a very detailed analysis and critical review of the fourteen cases published. He first very rightly throws out all those cases which were cured by the usual procedure during pregnancy or in which pregnancy could not definitely be held responsible, or which termination of pregnancy did not apparently help in curing. This throws out immediately Heermann's first four cases, Fremel's first two cases and leaves only eight cases to be considered. Similarly Bondy and Neumann's third case is disregarded, as it withdrew from observation before abortion and further treatment.

Blohmke denies that, as far as the cases reported are concerned, it has been sufficiently proven that pregnancy is the cause of acute exacerbations of a chronic purulent mastoiditis. He maintains that a reinfection from the outside is necessary and may take place without pregnancy. Similarly he denies that pregnancy has been proven to cause acute otitis media, as again one must have an infection from the outside, and these also occur without pregnancy. He further denies that pregnancy has been proven to retard or arrest healing in these cases, as such cases, either from the anatomic formation of the mastoid or from the nature of the infection, occur without pregnancy. He warns against too hasty radical operations, where only simple operation is indicated, for fear of causing premature birth; at the same time he cautions against danger of puerperal infection if a spreading process is not thoroughly cleared up.

He next brings up the question as to whether termination of pregnancy, either by normal labor or abortion, has been proven to have a beneficial effect on a progressing infectious process of the middle ear and mastoid. He claims that Neumann, Bondy and Fremel base the assumption that termination of pregnancy does this on the disappearance of the positive fistula symptom after labor and abortion. He claims that they do not know and have not proved that the fistula symptoms in these cases were real or false—whether a real fistula existed—and, therefore, cannot be considered as proof of this question. He further contends that rapid healing after termination of pregnancy proves nothing in these cases that healed slowly before term, as many wounds heal slowly for a long time and then suddenly heal over the remaining parts rapidly with no apparent cause.

Blohmke finally analyzes the question as to whether abortion is justified in cases of purulent otitis media during pregnancy in order to avoid aggravation of the ear condition. He does not consent to Bondy and Neumann's conclusions that abortion is justifiable to prevent exacerbations of a quiescent process or progress of an acute otitis media. He takes the stand that there is not enough evidence yet to make these conclusions.

Davis[15] also reviews Blohmke's work and cites a case of his own.

This is the literature and chief points of discussion on this subject to date. The questions raised were deemed of enough importance to justify the appointment of a committee[16] in the Viennese Otological Society, consisting of Alexander, Neumann and Stein, to collect more data on the subject from their confreres, in order to decide them.

My own case, reported in detail below, brought up the same questions to me, the patient's obstetrician and to the neuropsychiatrist we had in consultation, both of whom are leading men in their department at Stanford University Hospital. We were in ignorance of the few cases and literature on the subject, and it is only since then that I have found it has puzzled others before. As a case in point, it is, I believe, the most complete on record and as such justifies some conclusions on the above questions.

In this case pregnancy apparently, one might almost say definitely, repeatedly was an unfavorable factor in the progress of mastoiditis. It cannot be said that it caused the disease. On three different occasions I feel that it aggravated the condition and later retarded healing, and on termination of pregnancy the process definitely healed or was definitely benefited. On three different occasions the process progressed in spite of conservative and radical treatment to a place where grave complications threatened, and on these occasions the turn for the better came on the heels of the termination of the pregnancy, the first through spontaneous abortion, the second, Caesarean section and the third induced abortion. On the fourth occasion the mastoid had not broken down after two months of pregnancy, when the patient induced abortion, but the ear was beginning to cause some discomfort.

As regards abortion in acute or chronic middle ear infections in pregnancy, I do not believe we can generalize or make broad conclusion from the cases so far published. Each case must be individualized as it develops. If another such patient came to me tomorrow pregnant, I would watch her as before until such time as, in spite of treatment, she approached the danger stage. Then, and not before, I would certainly advise termination of pregnancy.

I do not believe that pregnancy as such is the cause of these conditions, but I do believe that every pregnant woman is carrying an extra load. Some can carry the pregnancy and in addition infections, nephritis, tuberculosis, etc. Others cannot carry even the pregnancy alone. That tuberculosis itself might be a factor in some of these cases was suggested in the discussion on Bondy and Neumann's cases,[12] but this was ruled out in my case by microscopic examination and guinea pig inoculation. If a patient has a progressing infection with her pregnancy she is carrying two loads, and if we may not remove the one load before it reaches the danger zone then we must sacrifice the other.

Case Report.—Mrs. A. J., age 21, first seen August 30, 1922, complaining of a discharge from her right ear.

History: At the age of 6 years two operations on right mastoid following scarlet fever, but discharge from ear continued off and on. At the age of 11 years right mastoid again oper-

ated on. This time process apparently cleared up, but has been completely deaf in this ear since. Has one child.

Present complaint: Right ear began to discharge two weeks following swimming two days before; menstrual period two weeks overdue. Since ear has been discharging she has had increasing shooting pains in the ear, deep parietal headaches with severe attacks of dizziness in which she almost falls.

Examination, August 30, 1922: Right ear: healed irregular depressed scar over mastoid. Small amount purulent débris and discharge in canal; total destruction of drum; promontory covered with unhealthy granulations. Pulsating discharge seen in moderate sized opening into attic. Posterior canal wall shows scars of some previous operative procedure. Spontaneous nystagmus of first degree to right and to left present. Fistula symptom pronounced, causing marked increase of nystagmus to left and marked vertigo. Left ear, as well as nose and throat show nothing of interest. Hearing: right ear, with Bárány's noise apparatus in left ear, watch 0, whisper 0, loud voice only when spoken into ear. Left ear, watch 3 meters, voice greater than 5 meters. R. B. C., 4,250,000; W. B. C., 10,800; polymorphonuclear, 68 per cent; lymphocytes, 29 per cent; large mononuclear, 3 per cent; hemoglobin, 80 per cent. Urinalysis, faint trace of albumin, no casts, a few leucocytes.

Radical mastoid operation, August 31, 1922: Scar tissue of old operation removed. Large pocket filled with granulations, mucopus and débris found in region of old antrum, walls of which were necrotic. Some cells in this region were involved and were cleaned up with curette and chisel. Granulations of middle ear and eustachian tube lightly curetted. No fistula into the labyrinth seen at operation. Small posterior pedicle flap turned in from posterior border of wound; wound closed with metal clips. Iodoform dressing through external canal.

For first six days following operation patient had marked nystagmus and vertigo on moving head on pillow and when dressings were changed near promontory and semicircular canal; temperature normal. On September 10, 1922, began to menstruate. Period was normal except for some larger blood clots than usual. Patient realized this was probably a miscarriage but did not mention it until later. This period was almost four weeks late. On September 14, 1922, very little

secretion present; the patient has been up for three days, although she was dizzy and nauseated yesterday (September 13th). Touching the region of promontory with cotton applicators caused marked vertigo and nystagmus. Left hospital on twentieth day.

September 25, 1922, small fistula seen near posterior limit of promontory, which pulsates, and when touched with cotton applicator causes marked disturbance, roaring and vertigo. Fistula balloons slightly with Siegel otoscope. September 29, 1922, epidermis rapidly spreading, fistula appears smaller. October 23, 1922, wound healed and dry. Seven weeks after operation patient went home to the country and was seen about once a month until May 7, 1923, during which time the wound remained healed and dry. She was well except for occasional dizziness on quick movements of the head. May 14, 1923, still has some dizziness on quick movements and some headaches at times. Some débris posterior to entrance, with a little granulation tissue which was curetted out. Still has spontaneous nystagmus of first degree to right and left in extreme position. May 21, 1923, caloric right ear caused very prompt rotary nystagmus to left. Past pointing both arms four and three inches to right. X-ray examination: "Irregular and not very dense area close behind auditory canal, which appears like spot of persisting periostitis." May 24, 1923, as the ear was healed and dry, I decided to disregard the X-ray findings.

August 24, 1923, ear has been dry until the last few days, when patient again has some secretion. Area just behind entrance to canal again shows some granulations and is very sensitive. Headaches very severe with some dizziness. Promontory region appears in good condition. Patient should have had normal menstrual period two weeks ago. August 29, 1923, increasing discharge with increasing severity of headaches and vertigo. Blood count of 12,000 W. B. C., 69 per cent polymorphonuclear, temperature 99°. Urine showed slight trace of albumin. Neurologic examination by Dr. Henry Mehrtens showed nothing on which to base a diagnosis of intracranial complications, such as brain abscess, although from the severity of the symptoms one might be suspected.

August 29, 1923, operation: Revision of mastoid, and at this operation the bone area behind the entrance to canal was found

necrotic, and this extended inward to well up to and posterior to horizontal canal. All affected bone and granulations removed. The postoperative course in the hospital was less severe than after the first operation. She was in the hospital only nine days. The outside incision healed normally, but the operated area in the mastoid trough did not heal. There was constantly a production of granulations and small rough bone areas could be felt.

Report from pathologic laboratory on specimen of tissue removed at operation states: "Infected granulation tissue, no evidence of tuberculosis found in the sections." Guinea pig inoculations were made from purulent material taken at operation, but the animals failed to develop tuberculosis.

The patient returned to the country and was to report twice a week, but between October 11, 1923, and January 31, 1924, she did not report. During this time she had diphtheria, which caused no change in the ear condition, which continued to discharge, and she still continued to have headaches with some dizziness. She reported on January 31, 1924, at which time there was very little discharge. She did not report again until March 19, 1924. At this time she stated that her headaches had become worse until they were unbearable; that her dizzy attacks were more frequent and severe. She was in her eighth month of pregnancy. Examination showed granulations in back of vault of mastoid trough, which when curetted away showed a deep opening under the epidermis of the trough, leading to rough bone behind and lateral to labyrinth. Treatment and observation during the next seven days did not improve symptoms or the condition of wound. There was a second degree nystagmus to left at this time. I referred the patient to Dr. Karl Schaupp for obstetric examination and explained the condition to him and that mastoid operation was again necessary. As she was so near term he consented to be present at the operation, in case the operative work should start labor pains.

March 29, 1924, operation: At this operation the area under the granulations was again cleaned away and necrotic bone again found on the posterior part of trough just lateral to labyrinth. The dura was found exposed over a small area. This area was carefully examined and then widened over an area of

about 1½ to 2 cm. The dura appeared normal. This time I did not close the posterior incision but left it wide open with the intention of keeping it so. Between March 29, 1924, and April 7, 1924, the patient suffered so greatly from both headaches, vertigo and nausea that only a negative neurologic symptom kept me from feeling that she must have a brain abscess. Dr. H. Mehrtens examined her neurologically and stated that, although she might have an abscess, still there were no localizing signs to base it on. On account of her severe head symptoms and on account of the fact that the dura had been recently exposed, I advised a Caesarean section be done rather than allow her to go through labor, and to this Dr. Schaupp agreed. On April 7, 1924, she began to have definite labor pains and Dr. Schaupp performed a Caesarean section that evening, an 8½ pound boy being delivered.

April 8, 1924. The day following the Caesarean section there was immediate cessation of headaches and dizziness and pain in ear. The wound healed very well except for one small spot behind and lateral to labyrinth, which did not heal.

October, 1924, she again reported that she was having severe headaches and dizziness again for a week or more. I immediately asked her if she was pregnant, and she said she should have had a menstrual period a week ago. Examination by Dr. Schaupp showed that she was pregnant. We decided to watch her for a few weeks. The ear condition became decidedly worse in spite of local treatment, the headaches and dizziness increased, and in consultation with Dr. Schaupp and Dr. Mehrtens it was decided that termination of pregnancy was advisable. Dr. Schaupp performed a curettement on November 10th, followed by immediate cessation of head symptoms and with immediate and complete healing of the wound. The wound remained healed for six months with no symptom of discomfort or breaking down. On May 10, 1925, the patient reported she was having some dizziness and pain and that her last menstrual period failed to occur a week previous. She went on through two months of pregnancy, during which the mastoid wound did not break down. From the beginning of this pregnancy she had increasing head symptoms of pain in and around the mastoid, with dizzy attacks and occasional nystagmus of first degree to the left. Part of the

head symptoms were neurotic, I feel sure, as she was very apprehensive of the ear complication. She went away to the country, and on July 1, 1926, caused an incomplete abortion. This was followed by a severe pelvic infection, which almost ended fatally, and necessitated later curettement and later total hysterectomy. Since this last pregnancy there has been no further trouble in connection with the ear or mastoid, which has remained perfectly healed. Although practically totally deaf in this ear, she has a perfectly functioning labyrinth, as shown by caloric irrigation.

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LXXXVI.

VISUALIZATION OF SINUS DRAINAGE.*

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If it were possible to observe accurately the spontaneous drainage of each individual sinus before operation, much needless surgery could be avoided. While this is obviously out of the question, a situation closely approximating the natural conditions can be brought about with the use of lipiodol, and the spontaneous evacuation of this oil can be observed by skiagraphy.

Granted that in some of its characteristics the oil differs from the secretions in question, which may introduce a variable element of error, still in viscosity and specific gravity the oil, as we use it (lipiodol and olive oil, equal parts), closely resembles the exudates, and the two fluids behave much alike in their passage through constricted openings, through small tubes and down moist inclined planes.

If our conclusions are to be of any value, the mucosa must be disturbed as little as possible in the process of introducing the oil. Cocainization is out of the question, as the initial overpatency of the ostium following its use produces an increased early drainage and the later turgescence an adventitious retention. This precludes injection through any puncturing instrument. Manipulations also with canulæ in searching for the normal openings often traumatize the tissues or set up vasomotor reactions sufficient to vitiate the results. Those cases in which the ostia are visible, or readily encountered, are seldom the ones which require investigation.

Instillation by displacement[1] has been found to meet the requirements, any slight hyperemia which it may occasion be-

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ing very transient, as results indicate. The lipiodol is introduced by the usual displacement technic,[2] and the relative filling of the cells is observed on the X-ray films taken at this time. Any anatomic or gross pathologic variations may be seen on this first film. If further skiagraphs are now made at regular intervals, the facility, relative and absolute, with which a given cell empties itself can be accurately determined.

We have here, it seems to me, a reliable index of the spontaneous drainage of each cell, and clinical observations strengthen this assumption. From a diagnostic—and prognostic—standpoint, the information afforded has been far more useful than the topographic data of simple opaque injection.

Sphenoid and ethmoid cells assumed to be healthy are found in our short experience to empty themselves completely in 96 hours. The rate of evacuation describes a rapidly diminishing curve. Twenty-four hour plates show approximately one-fourth of the oil still in place; this irrespective of anatomic variations in size, contour and position. A sphenoid cell with a pneumatized pterygoid process, for instance, empties itself as promptly as its twin of ordinary proportions.

The 48 hour plates show a decrease of about one-half of the previous day's oil shadow; on the 72 hour plate a mere flecking remains; the 96 hour plate may still show a trace or be perfectly empty. I should consider it inadvisable to lay down any arbitrary limits for surgical guidance. Of far more import is the relative drainage of a cell compared with its fellows. Furthermore, a sinus which drains itself in average or less than average time merits at least a reprieve, so far as operation is concerned.

Normal frontal sinuses empty themselves somewhat faster and maxillary antra somewhat slower than sphenoids. Again I refrain from fixing any limits. The actual time varies with the patient's sleeping posture, nose blowing habits and similar influences which must be considered. It is submitted, however, that these same factors which influence the evacuation of oil operate similarly in regard to exudates and therefore introduce no further source of error.

The routine followed at present is to half-fill the sinuses in question and to make the usual lateral and posteroanterior

skiagraphs with the X-ray beam always in the horizontal position,[3] and the plate vertical. The oil shadow produced by the usual vertical beam on the horizontal plate is so prone to variations brought about by mere anatomic conditions that we consider it unreliable and have abandoned that position.

Another exposure is made after 24 hours, in the position shown by the first day's films to be best suited to the study in hand. If any oil remains (as it should), a third and sometimes a fourth exposure are made at 24 hour intervals. From atrophic noses all oil may disappear in a few hours. Ostia which are partly occluded may permit the entrance of some oil under suction, presenting what might be interpreted from the first plate as a normal filling, and still prevent the spontaneous release of the oil for indefinite periods.

The patient, during the period of investigation, is given no instructions further than that he is to refrain from using any special manipulations with regard to his nose. He may blow it if he likes and should continue with his usual occupations.

When we begin to apply the information thus gained to the question of surgical procedure, elemental questions confront us: Is any benefit to be derived from an operation beyond the obvious and vital ventilation and drainage? If a cell rids itself completely of five or six mils of viscous oil in a few hours, is it not logical to assume that ventilation and drainage are adequate? What, if anything, is to be achieved by widening the opening of such a cell?

I do not know. These questions have been put to men of wide experience; the wider their experience the more guarded their replies.

In examining our series of films, one is struck with the frequent pneumatization of the root of the pterygoid process of the sphenoid. At first glance this condition appears to hold little more than anatomic interest until we recall that the narrow ridge which separates the main cavity of the sphenoid from this auxiliary one houses the vidian nerve.

Schaeffer,[4] in describing such a cell, has this to say: "In many of the specimens studied the floor of the sphenoid sinus is

separated from the pterygoid (vidian) canal by a fairly thick layer of compact bone. However, there are a large percentage of specimens in which the sphenoid sinus extends its pneumatization caudally and ventrally into the root of the pterygoid process. Indeed, the extension may pass beyond this, either medially or laterally or both, into the plates of the pterygoid process. In such instances the pterygoid canal, with its contained vidian nerve and vessels, is at best merely separated from the mucous membrane of the sphenoid sinus by an extremely delicate layer of bone. Indeed, in the majority of such cases the canal is thrown into marked relief in the floor of the sphenoid sinus so that it forms a distinct watershed dividing a lateral from a medial pterygoid diverticulum or recess of the sphenoid sinus. Again, it is not an unusual condition to find pterygoid canals in which the thin layer of bone is actually wanting (dehiscences), thereby exposing the contained vidian nerve and accompanying blood vessels to the influences of the outside atmosphere or of an infected sphenoid sinus. The mucous membrane of the sphenoid sinus alone is interposed between the sinus cavity and the vidian nerve, etc." The condition is clearly shown in the illustration from Schaeffer's text.

In 44 consecutive examinations I have encountered pneumatization of some part of the pterygoid process eleven times —exactly 25 per cent. Doubtful cases were classified with the predominating type. In only one case was the condition bilateral. (Fig. 7.)

Sluder, as early as 1910, studied the relations of the sphenoid sinus to the vidian nerve, and has repeatedly elaborated upon them in his later writings.

The clinical importance of these relations cannot be overestimated. Through the opaque visualization of the sphenoid alone, can they be adequately demonstrated in the patient. The topography of the region is such that simple X-ray exposures fail to throw it into sufficient relief for identification. On posteroanterior films showing pterygoid filling, the fluid in the sphenoid proper is an index of the prominence of the pterygoid canal, and therefore of the relative exposure of the vidian nerve.

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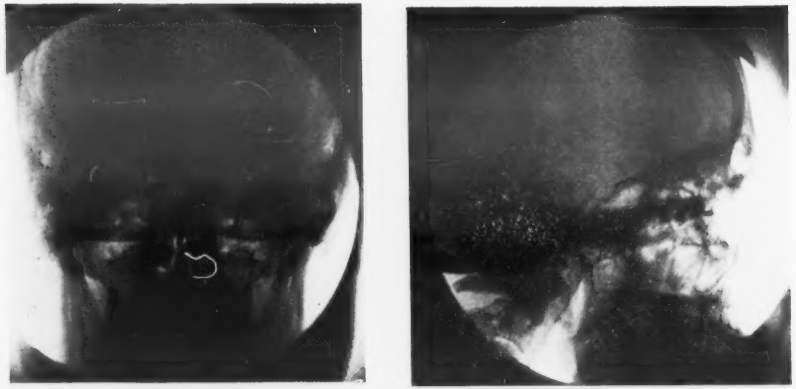


A B

Fig. 1. First plate showing average filling of the posterior cells.
A—Postero-anterior; B—Lateral.

Fig. 2. Twenty-four-hour plates.

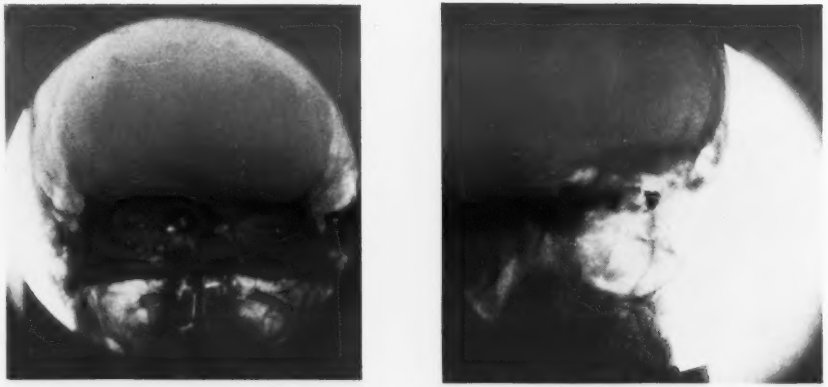


Fig. 3. Forty-eight-hour plates.

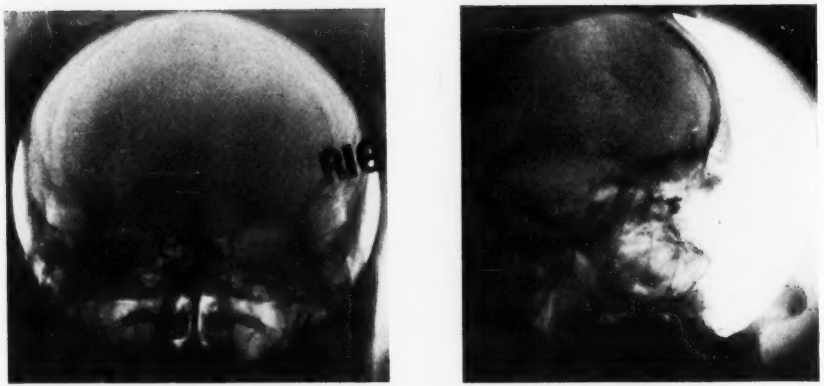


Fig. 4. Seventy-two-hour plates.

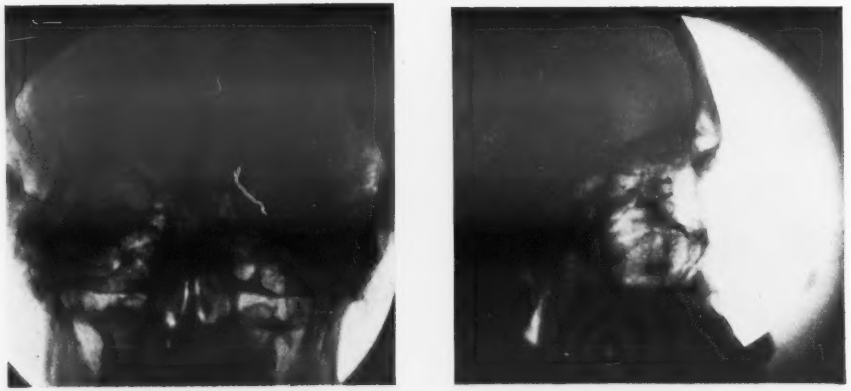
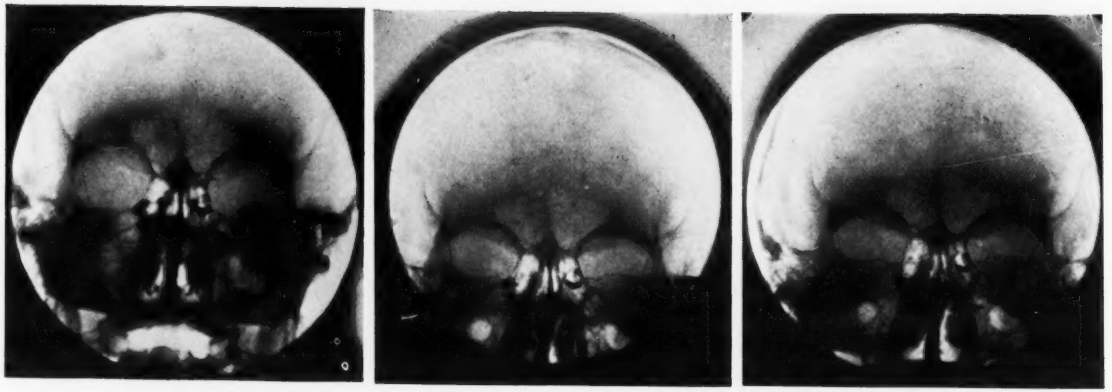


Fig. 5. Ninety-six-hour plates.



A B C

Fig. 6. Pneumatized pterygoid process which emptied itself in forty-eight hours. Case of retinal hemorrhage. A—First plate; B—Twenty-four-hour plate; C—Forty-eight-hour plate.

Figs. 7 and 8. Bilateral pneumatization of the pterygoid processes of the sphenoid.

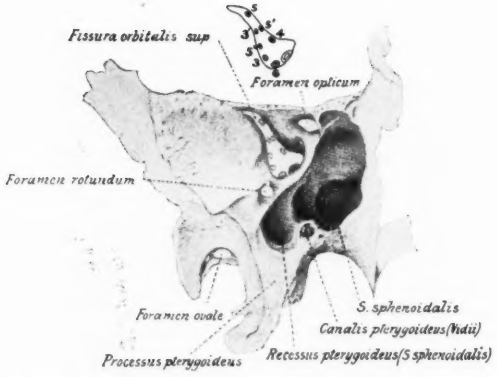


Fig. 9. Prolongation of the sphenoid sinus into the pterygoid process. Note exposure of Vidian canal (after Schaeffer).

LXXXVII.

PARANASAL SINUSITIS: A CLINICAL CONSIDERATION.

By John J. Shea, M. D.,

Memphis.

In these days of scientific medicine we are inclined to rely upon the laboratory for our information and to neglect the clinical study of our cases. If we study our own patients and observe their clinical courses, we will improve our diagnostic acuity and render to our practice better service. It is not the intention of this paper to cover the entire subject of paranasal sinusitis, but to limit our remarks to certain phases of the subject, some of which are accepted facts and others clinical observations.

ETIOLOGY.

Heredity.—A tendency towards sinus disease is often the fruit of the family tree. The frequency with which the children of parents suffering from sinus disease contract the disease early in life is a well known fact, and, irrespective of environment, diet or possible contagion, I believe that an inherited weakness towards the condition is a most important factor. This inheritance may be either direct or crossed, the latter being the more common. The sinuses of the child assume the same morphology as those of the parent or immediate antecedent whom it resembles in facial appearance, if the child and the parent have normally developed sinuses. This knowledge can be used to determine whether or not the child's sinuses are developing properly. We have rayed a number of parents with persistent infantile sinuses and have been unable to show that their offspring have a prototype of these dwarfed sinuses, and in our series we have had children with persistent infantile sinuses resembling in facial appearance a parent with a mature set of sinuses.

Bacterial Virulence and Selective Action.—This varies in each epidemic of upper respiratory infections. Likewise the

tendency towards sinusitis varies in the different epidemics, and at times the inflammation tends towards the formation of empyema.

The Influence of the Removal of the Tonsils and Adenoids in Young Children.—This is a subject worthy of the consideration of a special study, and, after careful observation, it is our opinion that children who have suffered the loss of their tonsils and adenoids at an early age are more inclined to succumb to sinus disease than children who have retained theirs until puberty. The logical explanation of this is that the sinuses play some part in the development of immunity in upper respiratory infections. The secretion found in an acute nose during the early stage of an inflammation is not only a physical protection to the membranes but has antibactericidal powers. When the tonsils and adenoid are in situ they aid in producing antibodies, and after their removal the sinuses are called upon to supply this deficiency. The ability of the sinuses is apparently limited, and in time they fall prey to the infection they are combating. In the study of 52 children below the puberty age who sought relief from symptoms suggestive of sinus disease I found that 35 had undergone operations for the removal of their tonsils and adenoid. Thirty-three of this number gave the history that their symptoms referable to the sinuses appeared after the removal of their tonsils and adenoid. The earlier their tonsils and adenoid had been removed, the more susceptible did they seem to sinus disease. Eight children in this series had lost their tonsils and adenoid, and contracted sinus disease before they had reached six years of age. I had removed the tonsils and adenoid of the majority, and personally observed the subsequent onset of the symptoms and signs of sinus disease in these children. In contrast, 17 children who had retained their tonsils and adenoid to their tenth year only eight had developed sinus disease. My attention was first attracted to this phenomena by parents returning with the complaint that since their children had been operated upon for diseased tonsils and adenoid, that with each subsequent cold the children would suffer nasal inflammations and would clear their throats and cough. The period between the operation and the onset of the nasal symptoms is, as a rule, two Winters. During the first Winter these children enjoy good health and

are free from disturbing infections, and it is my belief that during this period the sinuses are capably manufacturing sufficient antibodies, but, their ability being limited, the sinuses readily fail to carry on. The tonsils being inhibited by the gonads after puberty, are not thereafter such a power in autoimmunity, and their absence does not enter into the clinical picture. It is not my intention to discredit tonsillectomy during childhood, for I appreciate the wonderful results obtained in the majority of cases.

PATHOLOGY.

Discharge.—The character of the discharge, as observed after lavage, will aid in determining the stage of the inflammation. When the discharge begins to agglutinate, the stage of immunity is being approached. This fact further substantiates the theory that the sinuses are active in the process of immunity of the upper respiratory infections.

The Mechanism of the Development of Inflammation in the Sinuses.—This may be primary or by extension, the latter being observed when an empyema of one of the sinuses is the terminal stage of an acute infection of the upper respiratory tract. The former, when the first reaction to the infection begins within the sinuses.

Chronicity.—Each succeeding acute infection interferes with the nutrition of the membrane of the sinuses and their bony framework, and through faulty metabolism the process of repair is weakened. Seldom does the sinus become chronic from the initial infection, even though mild. Normally developed sinuses are less apt to become chronic than the persistent infantile type, because the latter have, through infection during their formative period, been arrested in their growth. The resulting hypertrophies of the nasal membrane and turbinates and the growth of polyps from recurrent infections by blocking the ventilation of the sinuses keep them in a state of chronic inflammation. Eventually the bony framework of the sinuses becomes chronic through faulty nourishment.

Vertigo.—Vertigo is a common symptom of acute frontal sinusitis, and rather constant whenever the patient stoops over and straightens up into the erect position, but its importance as a symptom in chronic frontal sinusitis has been neglected.

During the past year two patients who had been referred for the Bárány tests and an examination of the nose and throat showed in their roentgenograms shadows suggestive of osteoma within the frontal sinus, and a third referred for the same examinations showed a dental cyst which when pressed upon would excite an active vertigo. The latter phenomenon could not be explained. While experimenting with negative pressure in the sphenoid sinus I found that several of the patients at times would exclaim that they were having the same sensation as felt while diving. Positive pressure within these sphenoid sinuses failed to influence equilibrium.

Figure 1, Sugar Tolerance.—If a normal individual is given 100 grams of sugar his blood will show in a half hour its greatest concentration, which is, as a rule, 0.150, and by another hour will have reached the socalled normal of 0.100; but if the same amount of glucose is given a sinus patient it will pass the concentration of 0.150 and will require more than an hour to return to its basic level. If a diabetic patient develops a suppurative sinusitis, he will show a greater intolerance toward the glucose, and in one of our diabetic patients, with an acute frontal sinusitis, his blood sugar rose to 0.700 on what we supposed was a low sugar diet. With this knowledge that sinus patients have a low sugar tolerance it is my practice to remove glucose from their diet, and as they improve to allow of other "sweets" than sugar. Honey is the best tolerated of the "sweets."

DIAGNOSIS.

Roentgenology.—It has been stated before national societies that the knowledge obtained from roentgenograms of the sinuses is unreliable and of little value. This is the result of poor cooperation between the rhinologist and the roentgenologist. No set rule of timing can be followed in the taking of sinus roentgenograms, and often several sets of pictures are necessary to obtain the best results. We are taking today five exposures for the routine examination, (a) The "23° angle" by the Law technic, which gives the surgical relations of the ethmoids, frontals and antra; (b) the "chin nose," or Water's position, which demonstrates the floor of the antra; (c) a lateral, with the right side down, centered through the posterior

ethmoid cells: (d) the same with the left side down, and (d) a vertical through the vertex, which gives the best view of the sphenoids and the posterior ethmoid cells. The latter view also confirms the findings in the antra. In the past sinus pictures were made with too powerful penetration, and the result was a roentgenogram of the skull, and all of the sinus pathology was wiped out of the picture. In order to obtain the most knowledge, one or more of the views often are taken over with more or less penetration, as the case may demand. If the pathology is limited within the sinuses, the film should be soft, and if the changes sought are in the bony framework the film should be harder as for skull roentgenograms. In the examination of suspected malignancies the series should have at least three degrees of penetration—soft, average and hard. The first step in the interpretation of the roentgenogram is the determination of the stage of the development of the sinuses, for the sinuses reflect in their growth and development the infections they have suffered during their formative period. Fortified with this knowledge of their past history, the morphology of the right sinuses is compared with that of the left side. If in the Law view, the orbital shade is used as a standard for comparison, it should be slightly darker than the ethmoid or frontal sinus shadow. The latter two should be of the same shade. The roentgenogram that is the most often misread is that of the cystic type of ethmoids, for in this picture the ethmoids appear clear—in fact, they are too clear and signify that the membrane and intracellular walls of the ethmoids have undergone an atrophic change. If the pathology within the antra is pronounced and a better conception is desired, the sinus may be filled with lipiodol and the size of the membrane outlined or the presence of polyps proven. Proetz has given us an easy method of filling the sphenoid and posterior ethmoid cells with lipiodol. The antrum may be filled through a straight antral needle.

In order to obtain an average standard, I selected 100 roentgenograms of healthy individuals, free from sinus disease and representing the three periods of life. The roentgenograms were taken with the Law technic of 23 degrees angle, as this position renders the best anatomic picture. The sinuses of children under three years of age, as shown in these roent-

genograms, are represented by the letter A in Figure 1. The ethmoids extend up to the level of the cribriform plate; no frontal sinus shadow is present. The floor of the antrum is at a higher level than the floor of the nose. The average shadow of the cases at puberty are represented by the letter B, and have the following characteristics: (a) The frontal shadow is uniformly developed and its diameters are relatively equal; (b) the ethmoid shadow extends upward above the cribriform plate; (c) the antral floor is now on a level with that of the nose. The shadows in the adult roentgenograms may have a wide range of size and still be normal. The breadth of the frontal shadow should exceed its height, and the floor of the antrum is at a lower level than that of the nose. The shadow of the orbit measures relatively larger at birth than in the adult—in fact, its actual enlargement is immaterial from birth to maturity.

TREATMENT.

Ventilation.—The sinuses are air bearing cavities and are cured by ventilation and not by lavage. This is the reason why dental treatment of antral disease is a failure, for the dentist must depend upon lavage and cannot keep his opening into the antrum from being polluted by saliva and the contents of the mouth. Lavage is of service during the acute stage to rid the sinus of its byproducts of immunization which are toxic, but should be used as sparingly as possible. It is of little value in the treatment of the chronic infections. We are now convinced that the attainment of ventilation is the great value of allowing the rubber tubes to remain in the antral nasal wall during the postoperative week.

*Proetz, A. W.: A. M. A. Transaction, 1926.

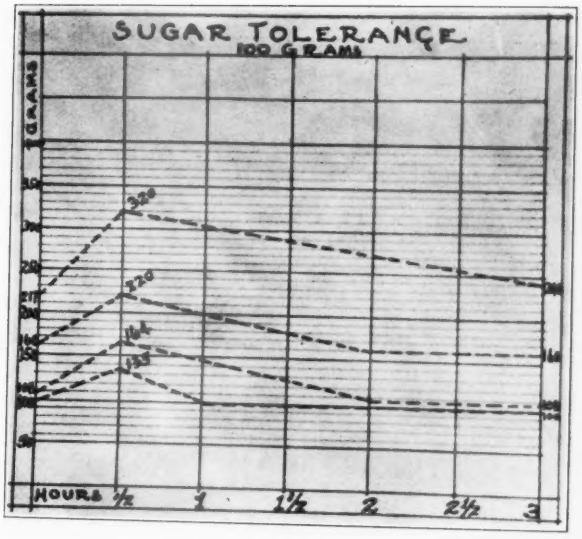


Fig. 1.

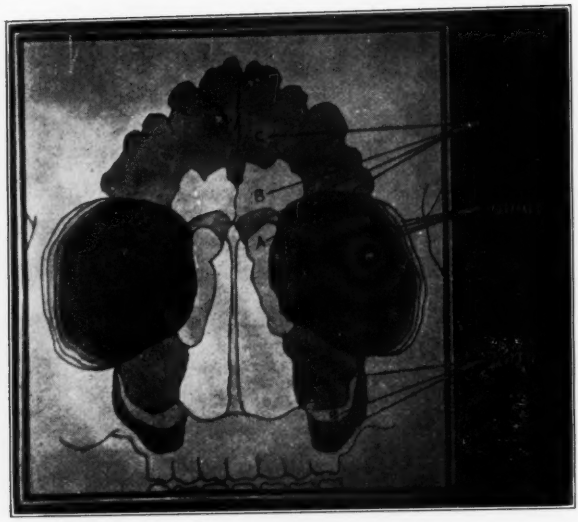


Fig. 2.

Fig. 3. Rubber tube in antrum for ventilation.

LXXXVIII.

DIFFERENTIATION BETWEEN OPHTHALMIC AND SINUS HEADACHES.*

By Ralph A. Fenton, M. D.,

Portland, Ore.

Fundamental to the discussion of headache should come brief consideration of the mechanism whereby this symptom becomes manifest. Confusion regarding classification exists in most clinical literature. "Headache" is made to include both internal ache from changes within the cranium and the meninges, and also neuralgic manifestations, local and referred, external to the brain and its envelopes.[1]

In 1860, Hilton's fourth lecture on "Rest and Pain" contained this suggestive admonition, as valuable nowadays as then:

"If the hidden cause of pain be in any one particular spot, it is only by tracing the nerves of and from that spot that we can hope to arrive logically at the real cause of the symptoms, and so divest the case of its obscurity. Applying this method to practice, it is through the medium of the distribution of the cerebrospinal nerves of sensation (the fifth nerve being the true cranial sensation nerve), that we are enabled to explain those pains which are called sympathetic but which result from a continuity of nerves between the cause and the effect, the disease and the symptoms."[2]

Bryce's reconstruction of a fifth week embryo (Fig. 1) sufficiently indicates the vast size of the Gasserian ganglion and the simplicity of its central situation with respect to the eye and the nose. But with the closure of gill slits, forward development of the cerebral hemispheres, anterior direction of the orbits, and relative arrest of development of the nasal and maxillary system in man, this early embryonal simplicity becomes a maze of complications. (Fig. 2.)

*Read before the American Academy of Ophthalmology and Otolaryngology, Detroit, September 12, 1927.

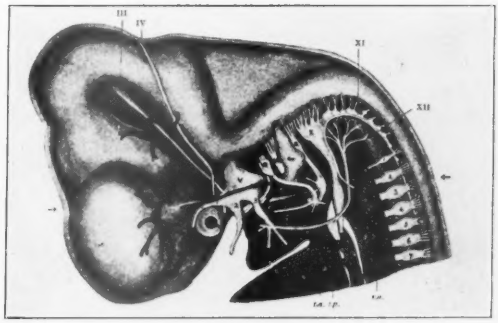


Fig. 1. Distribution of cerebral nerves and vessels in an embryo of about the fifth week. (Bryce, in Quain's Anatomy.)

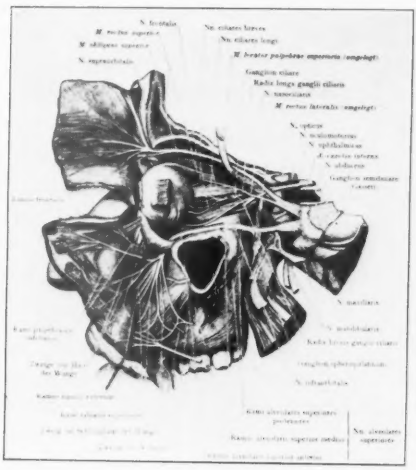


Fig. 2. Distribution of the first and second branches of the fifth nerve. (Toldt.)

Sensation from the eyes or the sinuses must travel inward over fifth nerve filaments for interpretation or for reference to distant terminals whose irritation confounds our diagnosis. Pain impulses travel from the Gasserian ganglion into the fifth nucleus; thence to the optic thalamus and the cerebral cortex.[3] Nerves of sensation are found in the dura, but have so far not been demonstrated in the arachnoid or pia. Regionally they are about as follows:[4]

Anterior fossa: Nasal branch of ophthalmic division of V. (especially over cribriform plate).

Middle fossa: Extensive branches from Gasserian ganglion of V. and mandibular nerve from V. (these extend up as far as the superior longitudinal sinus and communicate with the sympathetic plexus about the middle meningeal artery).

Tentorium: Recurrent nerve of Arnold from ophthalmic division V., near entrance to orbit, running back with trochlearis and ramifying inward to the straight, outward to the lateral sinus.

Posterior fossa: Recurrent branch from ganglion of the vagus IX. root, coming back in to dura via jugular foramen. Sensory branches from the loop between the hypoglossal and the first and second cervical to the posterior fossa and occipital diploe.

Vasomotor control and involuntary muscular action is vested in the fibers of the sympathetic system, which travel up from the superior cervical ganglion and along with the carotid plexus (Fig. 3), thence they accompany the cranial nerve trunks to their nasal and ocular terminations. These fibers traverse the sphenopalatine and ciliary ganglion without actual connection with the sensory fibers in these structures.[5 6 7] The assumption that sympathetic fibers carry pain impulses into the cranium and along the dura is not accepted. Nevertheless, such fibers may, if stimulated, readily produce localized changes in the dural circulation, with corresponding pressure and headache. Endocrin function, especially as to the sexual, adrenal, thyroid and pituitary secretions and their influence upon the sympathetic and its smooth muscle system, invite consideration here.

The socalled parasympathetic or autonomic fibers, which arise from the VII. and IX. nuclei and travel with the seventh

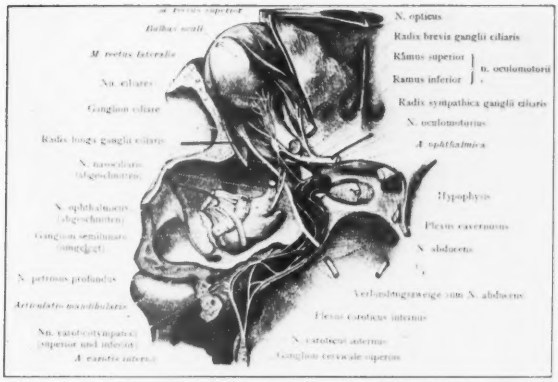


Fig. 3. Cephalic part of the sympathetic nervous system. (Toldt.)

and ninth nerves into the petrous bone, turn off within the pyramid to form the great superficial and great deep petrosals. (Fig. 4.) These join with a branch of the carotid sympathetic plexus to form the vidian nerve, running laterally to the sphenoid sinus and into the sphenopalatine ganglion from behind.

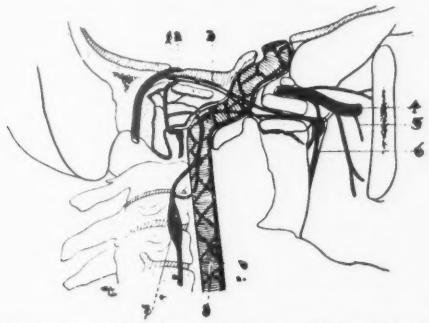
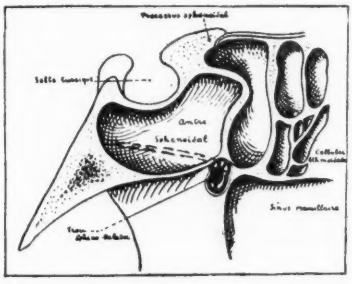
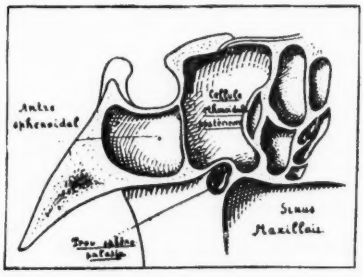


Fig. 4. Relations of the sphenopalatine ganglion with the facial and superior maxillary nerves and the sympathetic system. 1. Geniculate ganglion. 2. Facial nerve. 3. Great superficial petrosal nerve. 4. Superior maxillary nerve. 5. Posterior dental nerves. 6. Sphenopalatine nerve. 7. Cervical sympathetic. 8. Internal carotid artery and sympathetic plexus. (Terracol.)

From this point branches of the superficial petrosal accompany the orbital branch of the maxillary division of the V. to the lacrimal gland; others pass with the sphenopolatine nerve to the nasal mucosa.[5 8] (Fig. 5.)



5-A



5-B

Fig. 5A and Fig. 5B. Relations of the sphenopalatine ganglion area with the posterior ethmoid, the maxillary antrum, and the sphenoid. The course of the Vidian nerve in the lateral wall of the sphenoid is dotted into Fig. A (usual arrangement). Fig. B shows no relation between the sphenoid and the sphenopalatine ganglion. (Terracol.)

Afferent impulses through the autonomic fibers are carried back through the vidian nerve and the geniculate ganglion, and even on down into the cervical and upper thoracic ganglia. Herein we find explanation of referred aches in the ear, occiput and shoulder. These pain impulses are picked up by the posterior root ganglia and relayed on to the optic thalamus and

cerebrum, where they are perceived as originating in the ganglionic distribution rather than in the nose or cheek.[8]

The tough dural envelope (Fig. 6) is very sensitive to stretching and pulling. So accurate is the balance of the cerebrospinal fluid within the ventricles and cisternæ that we expect headache when this balance is disturbed by spinal puncture. Thus any change in the specific gravity of the fluid, any increase of contents, whether intravascular or hemorrhagic, cystic, neoplastic, exudative or otherwise, is bound to make pressure upon such branches of the nerves of sensation and

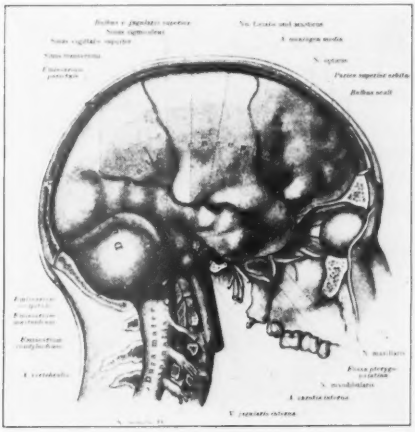


Fig. 6. Lateral view of the dura mater and cerebral venous sinuses in situ. (Toldt.)

vasomotor control as are found in the dura, and thus to produce headache.[9] Toxic substances and proteins (bacterial or specific), gaseous or liquid poisons acting directly or indirectly through the blood, are probably causative of headache through sympathetic effect upon the tonus of cerebral blood vessels, with consequent alterations in the formation of cerebrospinal fluid.[10 11]

Obviously, interference with free cerebral circulation by degenerative vascular changes, as in arteriosclerosis, angiomata,

aneurysms, postencephalitic adhesions, would render such a brain more susceptible to irritations from without.

Alterations in the pressure of the cerebrospinal fluid in the basal cisterna, whether due to change in the fluid or to interference with its circulation, are believed to cause pressure in the pain transmitting mechanism of the optic thalamus as to be responsible for the violent disturbances of socalled migraine. This "cerebral glaucoma" may cause lateral pressure on the optic tracts, accounting for coincidental transitory hemianopsia and scintillating scotomata.[12 13]

Let it be remembered that the sclera is an inelastic case comparable to the cranium, in that pressure upon its contained sensory nerves may readily be caused by changes in the fluid or chemical content of circulatory or lymph channels within the eye. This need not reach the stage of actual glaucomatous blocking of drainage, but may remain in the degree of active or passive ciliary congestion, or even of localized edema or relative hyperemia.

Let us be reminded also that the erectile tissue of the turbinates passes readily through every degree of congestion, from pale, waterlogged sogginess to the plethoric and hemorrhagic type.

The network of vessels in the nose and about the eye is accompanied by a far more delicate network of sympathetic nerve fibrils controlling vascular tone. Among these "vegetative" nerves are interspersed end organs of sensation which report mechanical pressure, edema, hemorrhage, passive or active hyperemia, as well as the results of pressure due to infection and inflammation, whether superficial or exudative.[6 14] Local toxins (bacterial, chemical, resultants of body wastes, fatigue or perverted metabolism) have similar irritant effects upon these sensory terminals.

In general, then, headache from these two fields is representative of local pathology, which is very often actuated through the sympathetic system, while it is reported to the region where headache is felt through the fifth nerve and its connections. Perceived within or about the cranium, it may, by reflex stimulation of the sympathetic at the perceiving area, set up local vasomotor disturbances within the dural envelope which may greatly aggravate the intensity of the original

headache. Conversely, conditions within the brain or along the sympathetic or sensory nerves may activate perceptory mechanisms within or about the eye or the sinuses, thus producing irritations or congestion which appear to be local.

With this fragmentary review of the anatomy and physiology of the nervous elements concerned in pain sensation from the eye and the nasal sinuses, we shall attempt to reconcile various clinical pictures reported by authorities in these fields. In extenuation of dogmatic statements, which through brevity sound rather too positive, we are reminded of a recent remark by H. G. Wells: "There is nothing so invigorating as a good generalization, but it ought to go through its facts and marshal them; it ought not to fly over their heads and expect them to follow."*

Sinus headaches, in the opinion of Skillern,[15] are due mainly to pressure, caused first, by obliteration of the sinus lumen by mucosal swelling, or second, by occlusion of the normal apertures with retention of secretion.[16] Socalled vacuum headache is in his experience uncommon, though it may be due to similar total occlusion of sinus ostia. Cystic turbinates or high thickenings or deviations of the septum, in so far as they obstruct sinus drainage and promote ethmoid congestion, favor the occlusive type of sinus headache. Bonnier of Paris, thirty years ago, attempted to prevent the sympathetic irritation which he considered causative of nasal headaches by cautery destruction of middle turbinal mucosa over the sphenopalatine ganglion. This foreshadowed Sluder's exhaustive study of sphenoethmoid neuralgias, which he attributes often to vacuum formation and to ganglion irritation, rather than to the end organ pressure of empyema or to turbinal and septal malformation.[17 18 19]

Skillern's classical diagram of the locations of sinus headaches is shown herewith. (Fig. 7.) Frontal sinusitis gives supraorbital to parietal pain; maxillary gives supraorbital;[20] the anterior ethmoid, at the inner canthus and supraorbital; the posterior ethmoid and sphenoid, parietal, postauricular and occipital.[5 21] Tabulation of the time of appearance of pain

*H. G. Wells: The World of William Clissold, N. Y., 1926, Vol. I, p. 162.

and of discharge is as follows—wide variations, of course, occur:

Sinus	Time of Pain	Discharge Observed
Frontal	8 a. m. to 1 p. m.	From noon on
Maxillary	Noon to night, or constant	On arising and all day
Ant. Ethmoid	Constant	On arising and all day
Post. Ethmoid and Sphenoid	Paroxysmal, often nocturnal	Variable; lateral pharyngitis

Excessive local tenderness above the inner canthus, on the thin floor of the frontal sinus, must be distinguished carefully from pain from pressure on the supraorbital nerve in its notch or from nasociliary neuralgia;[22] but this sign is helpful sometimes in diagnosis of unilateral frontal sinusitis. It should

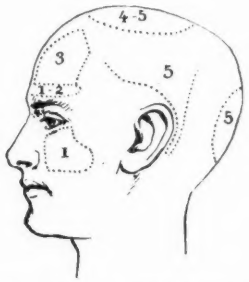


Fig. 7. Schematic illustration of pain areas due to sinus disease. 1. Acute maxillary sinusitis. 2. Acute frontal sinusitis. 3. Chronic frontal sinusitis. 4. Chronic ethmoidal inflammation. 5 Chronic sphenoidal sinusitis. (Skillern.)

not be forgotten, also, that many severe sinus involvements will have pain only on the occasion of an acute exacerbation, as following exposure, swimming and the like.[15 23]

Local factors in the consideration of ophthalmic headaches fall into three main groups: disturbances of motility, disturbances of refraction and accommodation, and disturbances of retinal function.

Protagonists of each group have claimed relief from such headaches through muscle operations and exercises; through cycloplegia and the correction of ametropia, especially astigmatism of very slight degree; or through physiologic rest and

the use of tinted lenses. Each group has many cures to report; but, as with the undiscriminating rhinologist, many disappointments have followed failure to consider all sides of the given case.[24 25 26]

Latent muscular imbalance is frequent, and its increase to manifest porportions is often an occupational hazard. Exophoria, being common, would seem to be frequently causative of headache. Hyperphoria, fortunately rare, almost always is productive of severe headache.[1 27]

Ametropia, whether it be in childhood or age, causes headache whenever use of the eyes is so exacting as to bring about congestion of the ciliary circulation owing to excessive or unequal use of the accommodative mechanism; or, in early presbyopia, owing to an attempt to force inelastic structures into action. Myopes rarely have headache; if present, it is due to the astigmatic component of their ametropia or to other pathologic features of the weakened globe. Irregular pull upon the ciliary muscle in cases of astigmatism, corneal scarring, keratoconus, posterior synechiæ, may often stir up most violent reactions without local manifestations in the eyes.[24 28 29]

Excessive demands upon accommodation from tasks at wrong distances must not be forgotten, especially in young hyperopes—factory workers, typists and comptometer operators and the like.[30] Weakened accommodation from illness, or specifically from the toxins of septic tonsils or teeth, will activate severe headaches.[1]

Retinal function, if attacked by excessive illumination, as by snowblinding, welding, switchboard flashes and similar intense discharges, is an obvious excitant for headache. Less obvious are the intoxications—alcohol, tobacco, quinin and others. Definite toxic agents, also working throughout the body, are found in acute or chronic septic states, in arteriosclerosis, in nephritis, in eclampsia, in neurosyphilis, in diabetes, in severe anemias, in socalled "acidosis."[31] Retinal sensitiveness, photophobia, itself an outstanding symptom in such disorders, may lead to headaches presaging the more usual pain of cerebral edema.

Far more common, however, is the dull ache of retinal fatigue, not alone from reading and writing, but in the world of industry. Typesetters, textile workers, machine feeders,

gem grinders, glass blowers, dental technicians, armature winders, thread cutters, stereotypers, are but a few of the multitude of specially trained artisans whose eyes may be emmetropic yet causative of headache from actual weariness of the overstimulated visual elements. Obviously general physical tire is a potent factor in such cases. Such patients report "all right after a vacation," or that "you can stand this job for about two years, then you have to quit and do something else."

Acute inflammatory glaucoma is a frequent and insidious cause of ocular headache, generally unilateral.[51 52] So frequently is it missed in the early stages that special consideration must be given to its quiet course and to its simulation of refractive asthenopia, retinal fatigue and the like. Ordinarily disease of the anterior segment should be fairly obvious: iritis, iridocyclitis, episcleritis and the like. Rarely, an overlooked foreign body, especially if entangled behind the iris or in the ciliary body, will stir up severe headache before local signs become manifest. The dull temporal discomfort and slight photophobia of a beginning sympathetic ophthalmia must not be ignored.

Ocular headaches are commonly frontal,[1 32] although certain heterophoria and ametropia cases complain bitterly of occipital and nuchal pain, perhaps from impulses relayed through the sphenopalatine ganglion and vidian nerve from the muscles and their orbital insertions.

Time of onset depends in general upon use of the eyes,[1 28 29] although severe fatigue or chronic disease may cause an eye headache to "hold over" and be present on rising in the morning.[29] Intrinsic causes, such as glaucoma and iridocyclitis, with their severe orbital and temporal "grinding" pain, are independent of the time factor.[28] The same is true of intracranial pressure conditions, with headache, which show eye symptoms. Thus cysts, neoplasms, gummata, aneurysms, abscesses, fractures, hemorrhage, localized meningitis, hydrocephalus and other cranial lesions may cause severe headache, accompanied according to location by choked discs, visual field disturbances, ocular palsies, nystagmus and other symptoms. Such eye findings are of course secondary to and in no sense causative of the intracranial pressure and corresponding headache.[9]

Congestive ocular pains, especially in glaucoma, are frequently accompanied by digestive disturbances and vomiting, so that much time may be wasted in gastric or general treatment before the eyes are suspected.[28 33 51]

Physiologic factors affecting our diagnosis include those distant connections of the sympathetic and autonomic systems which may modify the vascular supply of the eye or the nose.[34 35] Hypothyroid cases, with low basic metabolic rate, are frequently troubled with a nocturnal, occipitoparietal headache which closely resembles the sphenoid type. This, preceding menstruation, may be an indication of middle turbinate congestion; in fact, spontaneous nosebleed not infrequently occurs in such patients at these times.[36]

A full, throbbing, frontotemporal ache, with facial or bodily signs of giantism, and sliced down form fields above on the temporal side, suggest pituitary involvement rather than the frontal sinuses or sphenoids.

Pelvic congestion of long standing, with corresponding menstrual anomalies, produces headache by interference with vasomotor control rather than by direct interference with the ovarian secretory function. Debilitation from sexual excess will activate existing sinus or ocular headaches, just as any other extreme fatigue can do. No specific endocrine factor is at work in such cases.

Specific substances (foods, pollens, dusts) which cause angiospasm in the eye or nose may do so in the vessels of the dura. Local edema from such anaphylactic reactions may be very painful, or, in certain locations of the brain, may simulate very serious lesions of arteriosclerotic or hemorrhagic character. Diagnosis on such headaches is made by vasoconstriction (adrenalin, ephedrin) and by specific avoidance of the suspected protein.[36 37] An allergic basis for migraine, whether from bacterial toxins from intestinal stasis, or from certain articles of food, seems likely in many cases.

Chronic nephritis, diabetes, intestinal sluggishness and ptosis, cholecystitis, arteriosclerosis, lues, high anemia, leukemia and numerous other general disorders must be counted as factors of occasional moment in stirring up ocular or nasal headaches otherwise unimportant. The fundus will often fur-

nish a clue—optic neuritis, retinal hemorrhage, exudates, vitreous opacities, tortuous vessels, in such cases.[29 33 38]

Certain individuals are prone to magnify the importance of such a symptom as headache. Morbid psychology and the desire for sympathy may cause complaint long after the original cause of trouble has disappeared. In such cases fatigue type visual fields and red green reversal will often confirm the diagnosis of functional rather than organic disease.[1 39]

A careful history should, of course, precede any local measures of diagnosis. Often there is a family history of several members with recurrent headaches; this is especially true of heterophoria and ametropia. In the personal history, questions of occupation, use of the eyes, exposure, repeated colds or socalled "catarrh," are quite obvious lines of inquiry. Time of day or night, season of the year, will be indicative of sinus location, of allergic possibilities, of the cumulative effect of use of the eyes. In nocturnal headache, especially with a history of miscarriages, blood and spinal Wassermanns are suggested.

In examination to decide whether headache is ocular or nasal in origin, we must establish physiologic rest—freedom from local irritation or pressure—as a necessary preliminary. If the headache ceases under such investigation, we are on the right trail. Pitres[40] divides the pain from sensory terminals into four levels: (1) Peripheral pain, reached at the sensory ending, as by cocain locally; (2) pain stopped by anesthesia of the nerve trunk; (3) pain arising in the root ganglia of the spinal cord, as in tabes—this can only be reached by intrathecal anesthesia; and (4) pain arising from the medulla or cerebral cortex.

We may arrest sympathetic stimulation which is causing local congestion by adrenalin hypodermatically, ephedrin internally or ephedrin, adrenalin or cocain locally. We may deaden sensory endings in an affected area, thus arresting centripetal pain impulses and their cognate sympathetic stimulations at the more or less distant points to which the pain has been referred. We may place the eyes at rest by holocain and butesin locally, by cycloplegics or miotics, by dark glasses or prisms and by avoidance of near work and of artificial light.

Application of these methods may be carried out in a rather brief routine. From the history—colds, overuse of eyes or

what not—suggestions are likely to arise leading to definite lines of inquiry. Is the headache bad in the morning, better after noon? the frontals come to mind. With a forehead ache, are many handkerchiefs used, and is pus being cleared from the throat? the maxillary antrums should be investigated. Is there a sense of fullness between the eyes? consider the ethmoids. With pain behind the ear radiating to occiput and shoulder, we recall the sphenoid and posterior ethmoid. Visual field limitations, especially nasal, with deep boring temporal pain, suggest glaucoma. Bilateral frontal pain, late in the day, suggests astigmatism; if paroxysmal, or complicated by occasional occipital or parietal pain, heterophoria may be suspected. Transitory visual field losses, scintillating scotomata with dull parietal aches extending to the vertex, direct our attention to "migraine," with its implications of allergy,[37] of intestinal stasis, and of pressure in the basal cisterna.[12] Browache in middle aged people attempting much close work suggests increasing presbyopia.

Inspection will show us tired lines and wrinkles in the forehead, and crowsfeet and hammock lines in the lids, pointing to refractive error. Habitual head tilting, sidelong looks, even torticollis, may indicate muscular imbalance. Corneal scars or keratoconus, pigment deposits on the lens, scleral thinning are surface indications of extensive disease which may be responsible for refractive errors or for referred pain. Ophthalmoscopic inspection will disclose refractive errors, lens opacities, vitreous exudates, retinal edema, hemorrhages, exudates and pigmentation, arteriosclerosis, choking of the discs or the cupping of glaucoma.

Inspection in the nasal field should always precede the use of washes or shrinking sprays. One may thus detect swollen or cystic turbinates, perhaps impinging above against high deflections which may not impede breathing; septal thickenings equally obstructive of both sides; polypi in the middle meatus; the general direction of purulent discharges.[15 41] After shrinkage these items may be pursued further. In the pharynx lateral inflammation or supralateral pus accumulations will be noted.

For relaxation of strain from suspected refractive error, visual fogging by strong convex spheres may be tried. If

headache is thus helped, complete refractive measurement under adequate cycloplegia should be undertaken later. The cover test for latent muscular deviations is quickly done and should never be omitted in any dubious headache case. Frequently rather slight astigmatic errors will give rise to disproportionate pain. If relief be incomplete, trial prisms for near work, or convergence exercises, may solve the difficulty. Often neglected hyperphoria requires the red glass and Maddox rod for accurate determination.

Persons suffering from monocular amblyopia are less likely to have headache from muscular and fusion difficulties than are those with fair vision in both eyes.[42 43 51]

In the nose, inspection can only be completed after shrinkage by cocain, often with the help of adrenalin or ephedrin. Both middle turbinates should be examined, and cotton should be left in the middle meatus long enough to determine the presence of an outflow of pus, if not at first visible. Suction will occasionally cause pus to show itself; it may also give rise to a true vacuum headache, relieved by weak inflation. This may accurately reproduce the symptoms complained of by the patient. Anterior location of pus is usually frontal, but may also involve the anterior ethmoids: Pus in the posterior half of the middle meatus may be posterior ethmoidal, antral or rarely sphenoidal. Location of the pain and time of the discharge, as heretofore stated, must now be considered. In cases of closed empyema or mucocele, radiographs may be needed. In the posterior cells and sphenoids, the Pfähler or Rhese positions may be necessary. In such cases, lateral pharyngitis and postaural pain may be the only suggestions afforded the diagnostician. Transillumination of the antrum, if done as a routine, is as helpful as the radiograph, and just about as accurate. One is struck with the frequency with which "dark" antra producing frontal aches show no free pus on diagnostic irrigation, although there may be considerable dissolved mucin in the washings. Recent study shows the frequency of virulent bacterial involvement in these apparently empty antra,[44] whose thickened mucosa may be shown by lipiodol injection and radiographs. Lipiodol may be drawn after the method of Proetz[45] into the posterior ethmoids and sphenoids, to disprove the existence of closed empyema or to clarify the size and

relations of these cavities. Such pictures must be taken immediately after injection. Radiographs will sometimes disclose impacted molars or retained root fragments, which may cause neuralgia very similar to that of sphenoid disease. Dental pictures may not show enough pathology to account for antral invasion of marked virulence; nevertheless, it is wise to view "dead" teeth with suspicion, when frontal pain occurs on the same side. Incidentally, such latent dental infections are a potent cause of low grade iridocyclitis, with headache arising from difficulty in managing the inflamed accommodative mechanism.

Relief of congestion by bodily rest will commonly help both types of headache. Lying down quiets the circulation, and is often the only relief these people acknowledge. Jugular compression by tight collars or venous stasis in the head from bending over increase these headaches. Cold packs across the eyes will frequently allay the pain of refractive or muscular weakness or spasm, while that of frontal involvement may be rendered more violent. Hot packs may aggravate the pain of congestive ocular lesions, while affording some relief, apparently from transudation through the turbinates, in acute sinus disease. Many are similarly relieved by steam inhalations or hot saline irrigations. These findings are, however, most variable.

Relief of intracranial tension with corresponding headache is secured by strong saline purges, or even by strong saline injections intravenously. This forced dehydration secures immediate reduction of intracranial pressure, but it is a therapeutic rather than a diagnostic measure.[9 36]

Locally employed agents for decongestion include adrenalin, ephedrin and cocain. In accordance with the history and the local condition, we may shrink swollen tissues and do away with nerve pressure; then anesthetize the irritated areas and note the effect upon the distant pain. This is especially effective in the region of Meckel's ganglion. The period of relief following local use of dionin in the eye, after the first hour or two of severe chemosis, may possibly be a morphin effect, but to us it has seemed rather a decongestive effect by transudation.

In conclusion and recapitulation of these crowded, elementary observations, we are reminded of

The history, not forgetting family tendency, recurrence, exposure (weather, occupation, irritants), amount and kind of use (of eyes).

Based on the history, we look for nasal obstructions to drainage, injuries, malformations, septal and/or turbinal, inflammations, edema (allergic or toxic), neoplasms.

Ocular interferences with circulation and lymphatic drainage (corneal, iridociliary), inflammations (foreign bodies), increase of tension, edema (allergic or toxic), changes in the media, changes in the retina and nerve, muscular insufficiencies, refractive and accommodative errors.

The particular group of nerves which is irritated must be sorted out and set at rest, thus stopping or diminishing the headache, at least temporarily.

Such rest may be effected by: Local ischemia and/or anesthesia, avoidance of specific irritants, avoidance of irritating tasks. The headache may be central in origin, with symptoms referred to the eye or nose: relieved by general, not local measures: eye or nose symptoms are diagnostic of the general ailment; cerebrospinal, cardiovascular, gastrointestinal, renal. The headache may be psychic, with symptoms referred to the eye or nose: fatigue phenomena and unstable, irregular symptoms will be manifest.

We must not forget that ocular and sinus headaches may exist at the same time in the same patient. Nor should we forget the important rôle played by degenerative general disorders in building small amounts of ocular deterioration or nasal stasis up into relatively serious complications.[38 46 47 48 51 53]

No task in diagnosis is more difficult than the evaluation of all factors in these complex cases. Nowhere in medicine is an accurate history more valuable. With some searching of conscience, we may recall that S. Weir Mitchell, master healer of psychic pain, urged us, over fifty years ago, to beware of the easy alternative in diagnosis—hysteria or neurasthenia—and to look more carefully into the details of our own special fields.[49 50] Differential diagnosis can never rest upon headache alone.[52 53 54]

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LXXXIX.

SUPPURATIVE LABYRINTHITIS WITH REPORT OF CASES.

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The inner ear or labyrinth may become involved secondarily to middle ear suppuration in any one of several different ways. Karl Wittmaack[1] presents the most complete classification of the labyrinthine affections. The classification which he presents is based largely on the histologic changes found to have occurred in the inner ear of animals experimentally infected. He divides the inflammatory affections of the inner ear as follows:

1. Hydrops labyrinthi.
2. Serofibrinous labyrinthitis.
   a. Tympanogenous form of the serofibrinous labyrinthitis. rinthitis.
      Serofibrinous "window diffusion" labyrinthitis.
      Serofibrinous "capsular erosion" labyrinthitis.
   b. Meningogenous form of serofibrinous labyrinthitis.
   c. Hematogenous form of serofibrinous labyrinthitis.
3. The suppurative labyrinthitis and its sequelæ.
   a. Tympanogenous form of suppurative labyrinthitis.
      Suppurative "window diffusion" labyrinthitis.
      Suppurative "capsular erosion" labyrinthitis.
   b. Meningogenous and hematogenous forms of suppurative labyrinthitis.
4. Hemorrhagic labyrinthitis.
5. Necrosing labyrinthitis.
6. Specific forms of labyrinthitis, which are divided into
   a. Tuberculous labyrinthitis.
   b. Luetic labyrinthitis.

Alexander[2] discusses the pathologic anatomy of the inner ear according to the location of the lesion, after the following manner:

a. Pathologic anatomy of the labyrinth, meaning thereby the nonacoustic labyrinth.
b. Pathologic anatomy of the cochlea.
c. Pathologic anatomy of the osseous capsule and of the perilymphatic tissue.

According to the route of invasion, he divides them into the tympanogenous and the meningogenous forms; according to the character of the exudate into the serous (hydrops), serofibrinous and suppurative. The same author, in referring to the acute inflammations of the inner ear capsule, speaks of two types: First, the osteoperiostitic, which can develop from the extension of middle ear mucous inflammation or from the extension from the dural side; and second, the osteomyelitic, the latter originating and following the course of the blood vessels of the bone.

Very little consideration appears to be given to the subject of mere congestion of the inner ear, perhaps due to the fact that it is difficult to secure a case of the kind for anatomic study, especially since congestion of the inner ear is not in itself a sufficiently dangerous condition as to be fatal.

Max Goerke[3] classifies the inflammatory diseases of the inner ear according to the extent of the involvement into the circumscribed and diffuse forms (Figs. 1 and 2); according to the labyrinthine structures involved, into perilabyrinthitis, endolabyrinthitis and panlabyrinthitis. In the first mentioned the inflammation is limited to the perilymphatic space; in the second it is limited to the endolymphatic space, and in the third both the peri- and the endolymphatic spaces are involved. Certain toxins may show an elective affinity for the endolymphatic structures, causing an inflammation or degeneration of the sense organs located within the membranous labyrinth without at the same time involving the perilymphatic space. However, such an occurrence must be rare except in the case of meningogenous labyrinthitis, in which the infection spreads from the meninges, by way of the ramus cochlearis of the eighth nerve, to the ganglion spirale and to the ductus cochleæ.

The specimens which the writer has been privileged to examine, and also the illustrations presented by different authors, show the preponderance of pathologic changes in the peri-

lymphatic space. To the above mentioned three distinct pathologic processes involving the inner ear, Goerke adds a fourth, "paralabyrinthitis," in which the inflammatory process involves especially the labyrinthine capsule, leaving the membranous inner ear unaffected (Fig. 3).

The same author divides the inflammatory diseases of the labyrinth, according to the character of the inflammatory exudate, into the serous, fibrinous, serofibrinous, seropurulent and purulent forms. He also recognizes transitional forms which are difficult to classify definitely. Since the above classification does not exhaust every possible form of inflammation founded upon a purely pathologic basis, Goerke finds it necessary to add three other forms, according to the character of the inflammatory products; namely, the exudative, the plastic (Fig. 4) and the necrosing forms of labyrinthitis. The last mentioned is associated with sequestration of the labyrinthine capsule in whole or in part. (See Fig 5.) He recognizes, as do all who have given the subject any attention, the meningogenous as well as the tympanogenous form, according to whether the labyrinth is invaded from the side of the meninges or from the side of the middle ear. The latter is, of course, the more common.

The labyrinth is invaded in the tympanogenous form of inner ear inflammation through one of the windows (round or oval) or through a circumscribed loss of substance of the labyrinthine capsule. Uffenorde[4] gives credit to Steinbrügge, Habermann, Freidrich and others for having first shown by histologic findings that invasion takes place through the windows (Fig. 6); and to Marx, Mayer, Neumann, Herzog, Blau, Uffenorde, and especially Hayman,[5] for having confirmed the fact by experiments upon animals. Uffenorde claims that in twenty-nine cases of acute labyrinthine inflammation the invasion of the inner ear occurred twenty-seven times through the windows. The investigations of Schoetz,[6] Brock and Zange have convinced them that the infection generally takes place through the windows. Of the two windows the round one is generally accepted as the more frequent portal of invasion. Furthermore, in those cases in which both windows show evidence of having been the site of invasion, the round window reveals the more pronounced evidence (Fig. 1). Uffenorde[7]

claims that in 1910 he contended that it was possible for the labyrinth to be invaded from the side of the middle ear through the intact membrane of the round window. (See Fig. 7.) This form of invasion of the inner ear (dialysis of soluble bacterial toxins through the membrane) is referred to by others as "window diffusion" labyrinthitis. The resulting inflammation of the inner ear may not advance beyond the stage of a circumscribed serous or serofibrinous inflammation. It may, however, under especially unfavorable conditions, develop into the purulent form of labyrinthitis. The milder grade of inflammation results when the middle ear infection is a relatively mild one and the resistance of the membrane is strong.

In other cases the infection of the inner ear takes place through the window membrane only after it has suffered an open rupture. (Fig. 8.) The rupture may be single or multiple. It is impossible to tell clinically in every case of socalled "window invasion" whether the membrane of the round window is intact or not. Zange[8] showed, with the elastic fiber stain, that the elastic fibers are the most resistant constituent part of the membrane; for they were left when the membrane was otherwise completely destroyed by suppuration. The complete absence of blood vessels in the real fibrous membrane (membrana propria) makes that barrier stronger against invasion of the labyrinth by way of the round window, according to Uffenorde. The inflammation can, after the invasion of the inner ear through the window, come to a standstill, when in favorable cases there is partial or complete restitution of functional activity.

For our knowledge of the histologic changes in the tympanogenous acute labyrinthitis we are indebted to Herzog,[9] who conducted experimental researches on animals. In the case of "induced infection" there occurs early, in the region of the round window on the labyrinthine side, hyperemia, exudation and diapedesis of the leucocytes. Next there appears a homogeneous, cloudy, fibrinous like precipitate and cellular exudate. In most cases the inflammation spreads rapidly throughout the labyrinthine spaces. The involvement can, however, remain quite circumscribed, in that the intensity of the inflammation decreases more and more away from the window region. The sacculus in the case of an extensive inflammation generally be-

comes involved and to a marked degree. Herzog is of the opinion that the infection of the saccule takes place through the ductus reunions. The leucocytic content of the exudate and the inflammatory alteration of tissues depend upon the intensity of the irritant.

In addition to the window routes, the labyrinth can be invaded through a capsular erosion. The erosion may be merely superficial or may involve the entire thickness of the bone. (See Fig. 9.) In any event, it is possible for the bacterial toxins in the middle ear cavity to find their way through the erosion into the inner ear and produce a circumscribed labyrinthitis of the serous or serofibrinous type. This type of inflammation is comparable with the lower grades of meningitis (serous meningitis or meningismus), in which the exudate is serofibrinous in character and contains a relatively small number of round cells and very rarely polymorphonuclear cells. Such a character of inflammation can be produced not only from the extension of inflammations of the middle ear but from almost any kind of toxins, inorganic as well as organic.

The socalled "purulent capsular erosion labyrinthitis," according to Wittmaack, develops from uncomplicated "capsular erosion" labyrinthitis in exactly the same manner as the purulent "window diffusion" labyrinthitis from the serofibrinous. (See Fig. 6.) The only difference is that the transition from the milder form to the more severe takes place more readily in the case of "window diffusion" labyrinthitis than in the "capsular erosion" form.

The majority of cases of "capsular erosion labyrinthitis" heal up without reaching the purulent stage of inflammation. (Fig. 10.) Suppuration occurs only in those cases in which there is a local susceptibility because of the breaking down of infiltrations in the perilymphatic space due to lowered resistance from some systemic disease (arteriosclerosis, diabetes, nephritis and the like). The inflammatory process can also infiltrate the demarcation tissue and thereby provoke a purulent inflammation of the soft parts of the labyrinth.

Erosion of the labyrinthine capsule occurs most frequently in the exposed portion of the external semicircular canal (Figs. 11 and 12). Next in frequency is the footplate of the

stapes. It sometimes happens that multiple perforations occur at the oval window, for instance, one or more perforations through the annular ligament and another through the footplate of the stirrup. The promontory is not exempt. (Fig. 13.) Circumscribed resorption of the labyrinthine capsule from a growing cholesteatoma is not the same condition as a "capsular erosion"; however, it can bring about similar end results so far as involvement of the inner ear is concerned. In the case of cholesteatoma the process is somewhat milder; besides, its growth is sufficiently slow to allow time for the development of a productive type of circumscribed inner ear inflammation; affording thereby considerable protection against the invasion of the labyrinth by the more diffuse and destructive type of inflammation. In some cases the resorption of the capsule takes place clear to the endosteum, without the membranous structures showing the least involvement. (See Fig. 3.) This could not be so if the cholesteatoma or even its matrix were primarily infected. The cholesteatoma is composed, on its surface, of living cells. When, however, a cholesteatoma is poked with an unclean instrument, it can become infected, and in the presence of a fistula of the labyrinthine capsule can readily result in a widespread inflammation of the inner ear.

Purulent labyrinthitis as a pathologic entity is perhaps best described by Alexander.[2] Instead of discussing diffuse labyrinthitis as a whole, he considers the nonacoustic labyrinth separately from the acoustic; however, he recognizes the diffuse form when he discusses the acute suppurative inflammation of the cochlea. He divides the process into four stages: first, the initial stage; second, the stage of infiltration; third, the stage of purulent liquefaction (Einschmelzung), and fourth, the stage of healing.

The initial stage is recognized by the presence of fibrinous exudate, containing but few cells, in the inner ear spaces along with active hyperemia, circumscribed hemorrhagic areas, swelling and edema of the soft parts. Soon there develops mono- and polymorphonuclear cell infiltration which usher in the second stage. This infiltrate is seen early, and especially in and about the modiolus, the lamina spiralis, endosteal perilymphatic zone in the region of the stria and in that part of

the labyrinth which corresponds to the median wall of the inner ear.

Frequently the aqueductus cochlea is closed in consequence of the infiltration. There is swelling and infiltration in the region of the peripheral nerve endings. There is also some ectasia of the membranous cochlea. Staining for bacteria reveals the presence of microorganisms in the infiltrate.

The third stage, that of purulent liquefaction, leads first of all to cellular necrosis of the sense epithelium and of the nerve fibers, with their rapid and complete destruction. Profuse granulations are frequently found on the outer wall, especially in the region of the stria. The granulations are more profuse in the tympanogenous than in the meningogenous otitis interna purulenta. Subsequently the thin walls of the ductus cochlearis melt away completely, and eventually the more resistant parts—crista spiralis, membrana tectoria, ligamentum spirale—yield to the destructive influence.

The last, or the fourth stage, is that of healing, when first fibrin and later connective tissue envelops the suppurated parts and isolates them. The nonsuppurated parts of the exudate become resorbed in the remaining connective tissue, which eventually organizes into bone. In the case where the bony capsule is also involved and perforated with a fistula, the suppurated part is thrown off and emptied into the surrounding tissue. In the case of intact labyrinthine capsule, the suppuration runs an uncommonly long course. The intact walls of the membranous cochlea, through cicatrization and shrinking, become distorted, and the suppurated areas become organized into connective tissue and new formed bone. In this way, in many cases, the disease process is closed off. In other cases, however, without apparent cause, there follows an active growth of connective tissue or new formed bone extending in the direction of least resistance, toward the scalæ.

In this way the nervous structures, not already destroyed by the suppurative process, can disappear almost entirely by atrophy from pressure of the pathologic connective tissue. Finally, there follows obliteration of the inner ear through the filling up of its spaces with bone and connective tissue. Occasionally scar tissue or new formed bone will form parti-

tions within the labyrinthine spaces, the most important of which is the "vestibulocochlear septum" of Alexander.

Alexander recognizes a chronic form of inner ear suppuration. (Figs. 14 and 15.) He says the histologic picture is variously characterized. In some cases, through differences in the intensity of the pathologic changes, their recovery and healing take a long time, even when important parts of the inner ear have escaped the inflammation. In other cases the diffusion of the suppuration forms the principal cause of the chronic course. In most cases, however, the chronicity is due to recurrences of acute attacks of suppuration. Histologically one frequently sees in the same preparation recent and old inflammatory lesions side by side. To this is added the appearance of neoplasmatoid, connective tissue, and bony scars, and, at the same time, with middle ear cholesteatoma, the development of cholesteatoma in the inner ear. The eighth nerve atrophies sooner or later through the suppurative destruction of the nerve and its peripheral ganglion, in which the extensive necrosis plays the principal part; in recent cases, the secretion of hyaline exudate with formation of hyaline connective tissue. (Manasse.[10])

The most frequent source of purulent labyrinthitis is the tympanum. Just as in the case of the serofibrinous labyrinthitis, we must distinguish between the "window diffusion" and the "erosion" forms of purulent labyrinthitis.

The average case of suppurative labyrinthitis is probably preceded by the serofibrinous form with the characteristic changes described earlier. The serofibrinous form is due to the irritative action of the bacterial toxins generated in the middle ear that seep through the windows into the inner ear. With increased virulency, the infecting microorganisms break through the natural barriers and find their way into the inner ear when the serous form of labyrinthitis goes over into the suppurative form.

We are indebted to MacEwen[11] more than to anyone else for the explanation of the serous inflammation and its transition to the suppurative form, upon the basis of the former condition being due to the bacterial toxins that find their way to adjacent cavities by way of the perivascular lymphatics, the

latter condition being due to the bacteria themselves breaking through these natural barriers.

In practically all cases of suppurative inflammation it seems that it is preceded by the serous form. In some cases the duration of the serous form, or more precisely speaking, the serous stage, lasts much longer than in other cases. The interval of time between the onset of the serous and beginning of the suppurative form may be so short that the serous form or stage escapes detection altogether.

As to which course is taken depends upon the virulency of the microorganisms and the resistance of the individual. In those cases where the virulency is increased, by reason of poor ventilation and drainage of the middle ear spaces, the tendency for the serous labyrinthitis to give way to the suppurative form is greater than in those cases where the ventilation and drainage of the middle ear is improved, either by conservative or radical treatment. Again, the resistance of the patient figures considerably in these cases. In those individuals who are free of dyscrasias, the course is more favorable than in those who are not.

After a suppurative labyrinthitis is once established, the soluble toxins find their way into adjacent cavities and structures after the same manner as the bacterial toxins had earlier found their way from the middle into the inner ear; and, in those cases of very severe infections of the inner ear and in which the resistance is poor, the danger of intracranial complications becomes imminent.

The classifications of the inflammatory diseases of the inner ear pathologically and clinically differ in that the former recognizes physical changes, whereas the latter takes cognizance of functional deviations. One is just as important in its way as the other and both should balance.

The intralabyrinthine affections secondary to middle ear suppuration, capable of recognition clinically, can be divided according to the severity of the involvement into the irritative and the destructive; according to the extent of the involvement, into the circumscribed and the diffuse. The destructive lesions may be further subdivided, according to the length of time required to effect the loss of function, into the acute and chronic. However, it is practically impossible to tell in the

case of socalled latent labyrinthine suppuration, one in which there has been spontaneous recovery clinically, whether the pus that was present in the labyrinthine spaces during the active stage has since been ejected through a fistulous opening in the middle ear spaces described by Alexander[2] or has been retained in the inner ear and enveloped in scar tissue and new formed bone.

Where an active purulent infection within the labyrinthine capsule ruptures in any other direction than toward the middle ear cavity, serious complications are bound to arise. A detailed discussion of these complications will not be presented at this time.

It frequently happens that one pathologic condition of the labyrinth acts as the forerunner of another. In other words, one follows the other as a natural sequence. For instance, as a result of an especially virulent middle ear infection, in which the swollen mucous membrane, together with the products of suppuration, closes off the ventilation of the attic-antrum region, a fistula develops in the osseous external semicircular canal associated with a circumscribed congestion of the membranous canal, constituting clinically, therefore, a circumscribed irritative lesion. If the congestion reaches to and involves the crista ampullaris, the patient will experience some vertigo and at the same time manifest a spontaneous horizontal nystagmus directed toward the side of the affection. It is this combination of symptoms that suggests the character of the lesion; while the lengthening of the duration of the after-turning nystagmus toward both sides, especially toward the affected side, and the presence of a positive caloric reaction confirm the diagnosis.

A circumscribed irritative lesion once established may take one of several courses: (1) the process may become arrested, complete healing taking place organically with the restitution of physiologic function; (2) it may, in the presence of continued irritation or an increase in the intensity of the irritation, become worse and terminate in a circumscribed destructive lesion of the membranous labyrinth; or (3) it may, by reason of accentuation of unfavorable conditions (increased virulency of the infecting microorganisms or decreased resistance of the host or both), pass over into the diffuse form of inner ear suppuration.

Diffuse suppurative labyrinthitis manifests itself more often in the acute than in the chronic form. Clinically it is next to impossible to diagnose the chronic form; for, when the entire contents of the labyrinthine capsule are involved in a purulent inflammatory process the case will present all the symptoms and signs of labyrinthine destruction the same as is found in the acute form. It is true that the history of the case, if accurately presented, may suggest either a recent or long standing destruction of the labyrinth. For instance, when the patient presents the history of having gone through an attack of vertigo six months ago, severe enough to keep him in bed for several days, we may assume that there was an acute destruction of the labyrinth at that time, the more so, if he should add that he had not been able to hear from the suppurating ear since the attack. We cannot, however, from this history and with the additional confirmatory physical signs of labyrinthine destruction, tell whether the suppuration of the inner ear has healed or has continued as a chronic process. The presence of headache would lead us to suspect a continuance of the suppuration (chronic labyrinthitis), whereas its absence would suggest a comparatively early cessation of the suppuration with healing. There is no essential difference in the clinical manifestations of the otogenous labyrinthine suppuration and the meningogenous (craniogenous) form per se. The order of sequence, however, is different. In the former the intralabyrinthine manifestations antedate the intracranial, whereas, in the latter the order is reversed. Again, it must be remembered that the otogenous form is by far the more common.

The meningogenous form can readily escape recognition clinically, since it develops rather late, when the patient's condition is not favorable for conducting tests of the kind necessary to the making of an accurate diagnosis. On the other hand, there should be practically no difficulty in making the tests and arriving at a diagnosis in uncomplicated labyrinthine disease secondary to middle ear suppuration (otogenous form). This is especially true of acute diffuse labyrinthine suppuration in which the history and findings are classical. They are:

1. The history of middle ear suppuration of the acute or chronic form, more often the latter. The patient may tell us

that he has had a running ear for a number of years. Upon close questioning, the fact is brought out that the ear condition has been neglected. In a few cases of labyrinthine suppuration occurring in the course of acute middle ear suppuration the history of a running ear is not given, for the reason that the middle ear involvement at no time appeared to be sufficiently severe as to cause a rupture of the drumhead. Furthermore, upon otoscopic examination the drum head is found to be intact but not altogether normal in appearance. It looks rather dull in that the high light is absent. The landmarks are more or less preserved; however, one cannot detect the long process of the anvil as in the normal condition, because of the presence of an opaque secretion between the drum membrane and the anvil, which obscures the latter. An incision of the drum membrane will be followed by the escape of a mucoid or mucopurulent secretion which, when smeared and stained and studied under the microscope, reveals among other things the particular microorganism responsible for the infection. In these oddly behaving cases there is frequently revealed the presence of the streptococcus mucosus capsulatus.

II. Complete (Total) Deafness of Sudden Onset.—The deafness is complete. Many patients tell us that they are completely deaf when the functional hearing tests reveal the presence of a considerable remnant of hearing. In a doubtful case we must have some test upon which we can depend for determining whether there is a remnant of hearing or not. For a while the Politzer three meter speaking tube, in the hands of an expert, was sufficient for the purpose; but this gave way to the Stenger method with the double "A_1" forks of Bezold. The test was originally devised by Stenger for the detection of malingering. It proved so satisfactory that it was adopted by several members of the "Vienna Clinics," in 1907, as a test par excellence for determining unilateral deafness. It was at that time the writer first began making use of these forks in Alexander's Clinic. It was shortly thereafter that Bárány came out with his first noise producer, making use of a stream of water played against the drumhead. This soon gave way to his improved model which has proved so satisfactory since. The writer believes that he was the first to make use of the combined method of noise producer and speaking tube (in

1909), which is perhaps the least fallible of all methods. The method which he practices is to apply a two or three meter tube to one ear and the noise producer to the other. In the case of an ear with a mere remnant of hearing the patient is able to hear spoken words through the tube better than without the aid of the tube. Indeed he is often able to recognize whispered words. On the other hand, in the case of total deafness from destruction of the inner ear from any cause, including labyrinthine suppuration, words shouted directly into the mouth of the tube are not recognized by the patient. Furthermore, the patient presents quite characteristic fork findings, towit: Weber referred to the unaffected side; Schwabach decidedly shortened on the mastoid of the affected side; Rinne negative with no air conduction. However, all these fork findings put together are of less value in the detection of unilateral deafness than the combined speaking tube-noise producer test alone.

Total or complete deafness is not sufficient in itself to establish the diagnosis of acute diffuse labyrinthine suppuration, for we can find the same end results in the case of the chronic plastic form of labyrinthitis (Fig. 4), in which there has occurred complete metaplasia of the sense epithelium in the membranous cochlea. To check up exactly, the destruction of the hearing in the case of acute labyrinthine suppuration must have occurred suddenly. It should take no longer time to destroy the hearing completely than it does for a fulminating abscess once started to reach every part of the inner ear. It is not necessary for the superior cecum of the cochlea to undergo suppurative annihilation, for inflammatory edema will reach that far almost immediately, following the acute invasion of any part of the membranous inner ear with pus, and it alone is sufficient to produce a complete loss of function just as effectually as suppuration.

III. Vertigo.—The patient experiences the subjective sensation of turning in the frontal plane away from the affected side. At the same time, the outside world seems to be turning away from the affected side. The alert individual will speak of experiencing both of these sensations. The less alert will refer to one or the other but hardly both unless he is closely questioned. The sensation of turning is not confined to the

frontal plane but includes a combination of the frontal and horizontal planes. This is so, because in the case of diffuse destruction of the inner ear all three semicircular canals are destroyed and their combined influence produces the mixed horizontal and rotary form of nystagmus, which is directly responsible for the subjective sensation of turning experienced by the patient.

The intense vertigo of acute unilateral labyrinthine destruction develops just as suddenly as the deafness and for the same reason as given above under the heading "Complete deafness of sudden onset."

The vertigo of acute labyrinthine suppuration is more pronounced than that of an irritative lesion (congestion of the labyrinth) or a circumscribed lesion of the labyrinth, even of the destructive form. Furthermore, the vertigo of acute labyrinthine suppuration lasts for a much longer time than the vertigo of an irritative lesion. The vertigo of a suppurative lesion is most intense at the moment of destruction and decreases gradually thereafter.

After a week or so the patient complains but little of vertigo, for two reasons: First, the nystagmus which is responsible for the vertigo has decreased perceptibly by that time; and secondly, the patient has become more or less accustomed to the turning sensation, just as a whirling dervish or any other performer does whose act requires long continued turning.

The vertigo of an irritative lesion tends not only to be less severe than that of a destructive lesion but is also more transient, rarely lasting longer than a half hour. The vertigo of a circumscribed lesion, even of the destructive form, is also less intense than the vertigo of a diffuse lesion, for the reason that the direction of the sensation of turning is confined to a single plane, whereas the vertigo of a diffuse lesion includes the planes of all three semicircular canals.

There is one gratifying feature about the vertigo of a destructive lesion compared with that of the irritative one, and that is when the vertigo has eased up after a destructive lesion it never recurs, since it is not possible to further destroy a part already completely destroyed. In the case of an irritative lesion, on the other hand, recurrences of vertigo are the rule.

The report of a case of this kind was recently sent to Dr.

Goldstein for publication in the Laryngoscope. Briefly, it was that of a patient who suffered for a number of years with vertiginous attacks due to congestion of the inner ear, when finally it was decided to destroy the slight remnant of hearing she had by an operation upon the inner ear to relieve her of these repeated attacks of vertigo, which, indeed, were so severe as to take her "off her feet" on frequent occasions. The accumulated suffering of this patient from vertigo totaled many times that experienced by one with a destructive lesion of the inner ear.

The vertigo in labyrinthine destruction is increased when the patient looks toward the unaffected side and decreased when he looks toward the side of his lesion, for the reason that the responsible factor, nystagmus, is increased upon looking away from the side of the lesion and decreased upon looking toward the side of the lesion. In the case of an irritative lesion the vertigo is increased upon looking toward the side of the lesion and decreased upon looking away from it, because of the same controlling influence—nystagmus.

IV. Equilibrium Disturbance (Falling Reaction).—In the case of labyrinthine destruction the patient falls toward the side of his lesion, for the reason, that in his efforts to maintain equilibrium which he is falsely led to believe has been disturbed, he overcorrects and actually falls to the side opposite that to which he believes he is falling.

This falling reaction is manifested in other conditions in which aural vertigo is present. It is always in the direction contrary to that which the patient has been deceived into believing he is falling. The intensity of the nystagmus governs the intensity of both the vertigo and the equilibrium disturbances; for instance, when the patient looks toward the side of his nystagmus, the nystagmus is not only increased but also its concomitants—vertigo and equilibrium disturbance; and when he looks away from his nystagmus, the nystagmus together with its concomitants are decreased. It is for this reason that patients with severe vertigo, incident to a labyrinthine destruction, seek that particular recumbent position which tends to the production of the least intensity of nystagmus.

V. Spontaneous Mixed Rotary-Horizontal Nystagmus Directed Away from the Side of the Labyrinthine Destruc-

tion.—The nystagmus is rhythmic in character in that the movements are quick in one direction and slow in the opposite. In other words, the movements are jerky. The quick component is directed away from the side of the affection. It is not purely rotary in character, as one finds it in a circumscribed lesion involving one of the vertical canals, nor is it purely horizontal, as it occurs in a circumscribed lesion of the external semicircular canal, but it assumes the quality of both, since the lesion is a diffuse one involving all canals.

Although the nystagmus is very intense for the first few days, it begins to decrease from the moment it starts. After a few weeks it is barely perceptible on casual examination; however, it is possible to detect it upon close observation as late as a year afterward and even later. The writer recalls one case in which he was able to detect its presence several years after the destruction of the labyrinth from suppuration. To be sure, in these very late cases, the excursion of the nystagmus is very short (1 mm. at the equator of the eyeball) and the interval between the movements is rather long (six to eight seconds). However, it is very definite and unmistakable to one accustomed to observing nystagmus carefully. It is not desired to convey the impression that nystagmus never ceases in these cases, but rather that the spontaneous nystagmus as found in labyrinthine suppuration can be observed for a much longer time than is generally supposed.

If the four diagnostic factors thus far presented are found in the course of middle ear suppuration, there is nearly a hundred per cent chance that the labyrinth is involved in an acute diffuse suppurative inflammation.

The writer recalls that during the period between 1905 and 1908 there was a general feeling among otologists of the Vienna Clinics and outside that it was possible for a patient to present the clear cut picture thus far described and eventually recover with normal functions, when the condition was diagnosed diffuse serous labyrinthitis. This is the opinion of many otologists to this day. In a large experience covering many cases of labyrinthitis of different forms, the writer has yet to see a single case of this kind. He believes that diffuse serous labyrinthitis is rather a vague uncertainty. On the other hand, he accepts the possibility of diffuse serous labyrinthitis

associated with a circumscribed destructive lesion; but in this case there is no complete restitution of inner ear functions. Again, it is possible for a diffuse serous or serofibrinous labyrinthitis to result from the diffusion of bacterial toxins into the inner ear in the course of a middle ear suppuration; but again, there is no complete restitution of functions.

Although one may feel almost "dead sure" that a case presenting the evidence thus far produced points toward a labyrinthine suppuration, he is hardly warranted in operating upon this alone, until he has confirmed the diagnosis by certain important labyrinthine tests, except in emergency.

VI. Diminished After Turning Nystagmus to Both Sides but Especially Toward the Side of the Affection.—Normally the rhythmic nystagmus that follows turning is in the plane of turning but in the opposite direction.

After ten turns to one side with the head erect, there follows, in the average normal individual, a rhythmic horizontal nystagmus directed toward the opposite side, lasting for about twenty-four seconds.

In the case of unilateral labyrinthine destruction, the duration of the after turning nystagmus is found to be diminished toward both sides, but particularly toward the side of the destruction. In a recent case it seems to follow almost a mathematical rule, towit: the total duration of after turning nystagmus to the two sides is equal to one-half that found in the normal individual. For instance, if the normal duration of horizontal nystagmus after ten turns is twenty-four seconds to each side, it would total forty-eight seconds to both sides; while in the case of unilateral destruction, the total duration of the nystagmus to the two sides equals the half of forty-eight, or twenty-four seconds.

Again, the nystagmus is reduced two-thirds to the side of the affection, leaving but one-third of twenty-four seconds—eight seconds—as the duration of after turning nystagmus toward the affected side. The presence of this nystagmus cannot be due to any influence from the side of the destruction, but to the influence from the normal, opposite side, remembering that the semicircular canal of one side is capable of producing nystagmus to both sides, however, more so to its own than to the opposite side. Furthermore, the after turning

nystagmus toward the unaffected side in the case of unilateral destruction is reduced one-third of the normal—eight seconds —leaving the duration, therefore, two-thirds of the normal—sixteen seconds. These figures are fairly accurate for a recent case, but later on the difference in the duration of the nystagmus between the two sides becomes less marked. For instance, one month after a unilateral destruction the figures will change to about nine seconds' duration toward the affected side and fifteen seconds toward the unaffected. Again, the addition of these figures give the same result—twenty-four seconds. A month or so later the figures change to ten seconds' duration toward the affected side and to fourteen seconds toward the unaffected. The figures again total twenty-four seconds.

After many months the duration of the after turning nystagmus to the two sides will change to approximately eleven seconds toward the affected side and thirteen seconds toward the unaffected. After this stage is reached there appears to be no further change.

It will be noted that in the case of diffuse irritative lesions the total duration of nystagmus to the two sides is longer than the normal forty-eight seconds. It is this feature, more than any other, that helps us to differentiate an irritative lesion of one side and a destructive lesion of the other. For example, a patient with bilateral middle ear suppuration develops a spontaneous nystagmus to the right side, the question arises as to which inner ear is affected and what is the nature of the involvement. If the after turning nystagmus is increased appreciably above the normal to both sides but more so to the right than to the left, the right ear is the one involved and the lesion is an irritative one; on the other hand, if the after turning nystagmus is decreased to both sides but more especially to the left, then the left ear is the one involved and the lesion is a destructive one.

In spite of the fact that the turning test is a bilateral one, if we bear in mind the typical findings of the normal individual, there should be little difficulty in recognizing a pathologic condition and deciding whether the case is one of (1) hyper-irritability, from an irritative lesion, or (2) hypo-irritability,

from partial destruction, or (3) complete unilateral loss of function from suppurative labyrinthitis.

However, there remains an unfailing test that tells us in a case of hypo-irritability whether the destruction is partial or complete, and that is the test to be considered next.

VII. Caloric Test.—This is the most reliable of all tests in deciding whether the labyrinth is completely out of function or not. It is a qualitative test, pure and simple, in contrast to the turning test, which is a quantitative one. Each test has its own particular value, but both together are of more value than either alone.

The caloric test has the advantage over the turning test in that it is a unilateral one, whereas the turning test is a bilateral one. The caloric test is best conducted with the patient sitting in a chair with his head erect and syringing the ear with cold water, which is preferred to hot. Normal individuals will develop a pure rotary form of nystagmus to the opposite side after syringing the ear with cold water. The length of time required for the production of this nystagmus depends upon the temperature of the water. The colder the water, the quicker the reaction will appear. However, excessively cold water, 45° or thereabout, might be dangerous. The writer recalls a case in which the patient was made quite deaf for a day or so following the use of very cold water. While making the test with cold water it is possible that the patient may develop a very intense nystagmus (three mm. length of excursion repeated from ninety to a hundred times per minute). Since the presence of a less intensive nystagmus from the use of cold water tells us just as much about the reactibility as the more intensive nystagmus, it is not necessary to allow the nystagmus to develop to the more pronounced degree. With this in mind and too the thought of sparing the patient as much as possible the vertigo that attends the nystagmus, the writer is satisfied the instant the typical reaction appears and begins to neutralize the cold water effect with hot water. The first effect of the hot water is to cause a diminution in the intensity of the nystagmus to the opposite side that was produced by the cold water. The continued application of the hot water causes a reversal of the direction of the nystagmus. The writer is in the habit of keeping at hand hot water ready

to use the moment it has been definitely determined that the cold water reaction is positive.

If, after a reasonable application of cold water, say eight or ten minutes, use of three thousand cc. of water at 65° Fahrenheit, no nystagmus is produced nor change in the existing nystagmus, one is justified in concluding that the reaction is negative. It is important before making the caloric test to know exactly whether the patient has spontaneous nystagmus or not, and if so the plane of the movements, the direction of the quicker component and the intensity; otherwise, one can readily fall into error. For instance, if a patient is manifesting a nystagmus toward the left side in the case of questionable labyrinthine suppuration on the right side, the application of cold water will produce no effect on the existing nystagmus if the labyrinth is totally destroyed. On the other hand, if there is a remnant of function remaining, cold water syringed into the right ear will increase the intensity of the nystagmus already existing. It is not easy, even for a careful observer, to decide whether a slight accentuation of the nystagmus, from the application of cold water, has occurred or not, for the reason that the accentuation, when it occurs, does so very gradually. Likewise does the diminution following the termination of the reaction take place very gradually.

The writer, when he first began the practical application of the caloric test, applied it not only with the patient's head erect, but with the head in the horizontal position and side of the face inclined toward the opposite shoulder. In this position a positive reaction of the semicircular canals is manifested by the development of long excursion horizontal nystagmus directed toward the side being tested. Conducting the test after this manner avoids the objections just raised concerning the test in the upright position. In other words, it is much easier for the examiner to recognize a change in the direction as well as the plane of the nystagmus. The information to be obtained from the caloric test is so important that the writer feels it is safer to use both methods rather than depend upon a test in which a change in the intensity of the nystagmus is the sole factor in determining whether the reaction is positive or negative.

It naturally follows that change in the temperature of the water from hot to cold or vice versa reverses the direction of

the nystagmus. The plane of the nystagmus is altered only by change of position of the head; for instance, with the head erect, cold water produces a rotary nystagmus to the opposite side; whereas, with the side of the face turned to the opposite shoulder a horizontal nystagmus is produced toward the same side. One should bear in mind that an obstruction in the external auditory canal in the form of a foreign body, exostosis or large polyp can prevent the water from reaching the median wall of the tympanic cavity and result in a negative caloric reaction, even in cases with a normal inner ear.

VIII. The Galvanic Test.—The reaction of the inner ear and the eighth nerve on the side of the lesion is diminished—that is, to say, a greater amount of stimulation, with the cathode, is required on the affected side to produce a reaction (mixed rotary-horizontal nystagmus) toward that side than is normally required. If four M. A. of cathodal current (stimulation) applied to the external auditory canal in a normal individual produces mixed rotary-horizontal nystagmus toward the side of the cathode, then in the case of labyrinthine destruction eight M. A. will be required to produce the same reaction. This reaction results not from the stimulation of the end organ in the ampullæ of the three semicircular canals, since they are destroyed in the suppurative labyrinthitis, but from the nerve centralward to these end organs. The nerve stem is still capable of stimulation with a cathode.

The application of four M. A. of anodal current to the ear of the average normal individual produces a slight but perceptible mixed rotary horizontal nystagmus to the opposite side. In the case of a labyrinth destroyed from suppuration with an intact eighth nerve and the patient manifesting a mixed rotary-horizontal nystagmus toward the opposite side, the intensity of the nystagmus will be increased perceptibly with the application of the least amount of current strength (one M. A. or less); thereby proving that the tonus centers are not confined to the inner ear, as one might be led to infer from the presence of spontaneous nystagmus toward the opposite side in case of destruction of the inner ear.

When the cathode is applied to the opposite ear, in the case of labyrinthine suppuration, it will cause the existing nystagmus to be increased with a weak current strength (one M. A.

or even less). This is due to an increase of the total tonus to that side (normal tonus, which is equivalent to four M. A. plus the additional stimulation of one M. A.).

When the anode is applied to the unaffected side, four M. A. of current is required to arrest the existing nystagmus to that side and four more M. A., making a total of eight M. A., is required to cause a reversal of the nystagmus to the side of the inner ear destruction. These reactions, cathodal and anodal, on the two sides in the case of labyrinthine destruction, are typical so long as the vestibular branch of the eighth nerve is intact. In the case of destruction of the vestibular nerve the reaction is quite different. For instance, in neuritis, tumor of the eighth nerve or tumors of the internal auditory canal or any other pathologic condition in or about the nerve that is capable of destroying its conductivity, no amount of cathodal stimulation on that side will produce a reaction. In other words, it is not possible with the cathode applied to the side of the affection to cause a nystagmus to that side or change the character or direction of the already existing nystagmus. However, it is possible with the cathode applied to the side opposite the lesion to increase the already existing nystagmus just as in the case of a destruction limited to the inner ear.

IX. The Fistula Test Is Negative.—It could not be otherwise, even though a fistula of the labyrinthine capsule actually existed, for the reason that the end organs in the labyrinth have been so completely destroyed by the suppuration that it is impossible for them to be influenced by endolymph motion produced by the compression or aspiration of air in the middle ear spaces. This test is of little value as a diagnostic one in cases of labyrinthine destruction except where it is known that the fistula sign was present a short time (a few days) before. This last combination (fistula test previously present, subsequently negative) would indicate the presence of a fistula of the labyrinthine capsule with membranous labyrinth intact earlier and destroyed later.

INDICATIONS FOR OPERATION.

In the presence of a positive diagnosis of a diffuse labyrinthine destruction, secondary to a middle ear suppuration,

the question naturally arises, What are we to do in the case? Generally speaking, it would be safer in all cases of the kind to perform a radical mastoid operation, followed up immediately with an operation on the inner ear for the purpose of draining it. The reasons are as follows:

1. That these cases present, besides the evidence of inner ear abscess, all the symptoms and signs of middle ear suppuration sufficiently severe in itself to call for a radical mastoid operation, which if allowed to go unoperated, can terminate fatally from some other intracranial complication, secondary to or even independent of the inner ear abscess. In other words, a suppurative process of the middle ear severe enough to invade the adjacent inner ear could also invade the adjacent intracranial structures (the meninges, brain substance or venous sinuses). Besides, a suppurating inner ear is always a menace by reason of the facility with which the infection can spread along the preformed ways to the intracranial structures of the posterior fossa.

II. The risk to the patient's life from operating on the labyrinth is far less than from failure to operate. In fact, the risk from operating the labyrinth where the infection has not already spread to the posterior fossa is practically nil. Indeed it is no greater than in the ordinary radical mastoid operation.

III. The risk to the patient's life from operating upon the mastoid and middle ear spaces and leaving the suppurating labyrinth untouched is greatest of all. The writer witnessed two fatalities where the surgeons, both very capable men, had hoped that the loss of function in the labyrinth was but transient, due to what they supposed was merely serous labyrinthitis. They trusted the radical operation alone would suffice. Both cases died in less than forty-eight hours from fulminating purulent meningitis hastened by the incomplete operation of a suppurative labyrinthitis.

IV. In the case of a diffuse labyrinthine suppuration, there is no reason why anyone should hesitate about draining the pus from the infected ear. It is a logical surgical procedure and in itself can do no damage to the anatomic structures that have not already been destroyed in the suppurative process; remembering, too, that a suppurating inner ear can never function again, even without an operation.

OPERATION.

As otologists we should be concerned, first of all, in the accuracy of our diagnosis and the methods used in arriving at our findings. After the diagnosis has once been established, there should be no question as to the particular procedure that should be adopted.

The object to be attained in the presence of a suppurating inner ear is the most thorough and efficient drainage of the inner ear spaces possible, avoiding at the same time injury to the uninvolved adjacent structures.

The purpose of the present paper is not to go into the technical details as practiced by different surgeons, but to cite briefly the method the writer has employed for the last nineteen years. It is nothing more than a modification of the Neumann operation. The writer's modification, he believes, is the simplest of all methods, at least for him.

The radical mastoid operation is done after the Kuester-Bergmann method. The sigmoid sinus is next exposed and the posterior surface of the petrous bone is removed from the anterior margin of the sinus groove to the internal auditory canal, and from a line just above the inferior petrosal sinus to another line just below the superior petrosal. This must include, therefore, the posterior semicircular canal and the aqueductus vestibuli.

The superior surface of the petrous bone is removed anteriorly and medianward far enough to include the eminentia arcuata. This must necessarily include the greater part of the superior semicircular canal. The bone of the solid angle between the superior and posterior canals is next attacked, working lateralward, with a No. 4 Alexander chisel, toward the vestibule from behind and medianward.

The external semicircular canal, which was exposed at the time of the radical mastoid operation, is easily recognized. The upper half of this is removed to the vestibule, working from behind and externally, whereas Neumann's method is to attack the external canal before he does the verticals. The vestibule is entered next from in front of and below the facial canal, using a very small chisel (Alexander No. 4), uniting the oval and the round windows and the external aspect of

the cochlear whorls by removal of the thin plate of bone that comprises the promontory, care being taken not to remove the bone too high up. The writer wishes to emphasize this last point, since he feels that in at least one case subsequent facial palsy was due to injury of the seventh nerve at the geniculate ganglion, which is found very slightly above and anterior to the upper level of the oval window.

The operation is completed with a plastic, after the Panse method. The wound is treated after the open method, and a secondary closure made two weeks later in those cases where the opening does not show marked evidence of early closure. The healing is usually as rapid as after the ordinary radical mastoid operation, while the percentage of cases in which there is slight residual discharge is even smaller.

CASE REPORTS.

The following case reports are selected from a larger series, as those most suited for our present purpose. Some cases had to be omitted for one reason or another as not suited for reporting in this group. For instance, emergency cases seen with general practitioners in out of the way places, where the writer did not see the case again, and only learned of the final outcome by written correspondence, which lacked the proper sort of detail. In other cases the emergency was such as not to allow opportunity for the closest study; some, too, because of other complications; for instance, meningitis in which it was next to impossible to determine whether the labyrinthitis was primary or secondary to the meningitis. In still other cases the hospital facilities were exceedingly unfavorable for accurate study. The question might be asked why operate upon the labyrinth without indications of a most conclusive character? The answer is that there was present sufficiently strong evidence to satisfy the writer of the diagnosis, without at the same time affording those details which bring credit to the reporter. Yet something had to be done in such emergency cases, for to do nothing was more dangerous than to go ahead. For illustration, when called to see a case in a distant town where the only available hospital is poorly equipped; the patient lying in bed very much indisposed, and already presenting signs of beginning serous meningitis; no X-ray films of

the head having been made and no possibility of getting any within a reasonable time; the patient so sick and incapacitated from vertigo, vomiting and equilibrium disturbances as to prevent him from getting out of bed and permitting of a turning test in a chair improvised for the occasion.

In the presence of very pronounced mixed rotary-horizontal nystagmus directed toward the side opposite the affected ear; negative caloric test made after the manner suggested above; the characteristic finding of total deafness, with the combined speaking tube and noise producer, the writer feels that the chances are a hundred to one that the patient is suffering from an acute suppurative labyrinthitis, in which case it is safer to operate upon the inner ear and drain it of pus than not. Yet, for the purpose of reporting, the case lacks certain essential details. On the other hand, there is a slight chance that the case is not one of labyrinthine suppuration, after all. Such an unique case the writer hopes to report on some other occasion. It is one of left sided chronic middle ear suppuration associated with neurolabyrinthitis of focal infection origin.

Case 1.—Mrs. O. H., age 51 years, was referred by Dr. Wesley J. Barrett, of Camden, N. J. The patient was first seen by the writer on October 23, 1918. The history was that of left sided middle ear suppuration of three years' standing. She claims not to be able to hear at all on that side and has suffered a great deal with severe attacks of headache. After these "terrible headaches" the ear discharges quite freely and she feels very dizzy. There is pain in the back of the neck and "all the nerves on the left side of the face." For the last three months the pain in the head has been very intense. Her appetite is poor and she has lost thirty-two pounds in weight.

Functional hearing tests show but slight reduction of hearing on the right side; Weber referred to the right side; Rinne positive; Schwabach slightly shortened; slight shortening of hearing by air conduction for the low and middle tone forks; normal hearing for the high 2048 d. v. fork. On the left side there is complete deafness to air conduction for low, middle and high tones. With the three meter speaking tube in the left ear and the noise producer in the right, the patient is unable to hear words spoken into the mouth of the tube with loud conversational voice.

Otoscopic examination: Right ear reveals an intact drumhead, quite normal in appearance and behavior; left ear reveals the presence of pulsating purulent secretion which, when wiped away, allows one to see a large round perforation just behind the umbo. The mastoid is tender to finger pressure.

There is a slight degree of mixed rotary-horizontal nystagmus to the right side when the patient looks straight ahead. After turning nystagmus: After fifteen turns to the left, horizontal nystagmus to the right, fourteen seconds; after fifteen turns to the right, nystagmus to the left, twelve seconds. The caloric reaction is negative on the left side.

The patient was operated upon the same day, after the method already described—that is, the radical mastoid operation, followed up at the same sitting with an operation on the labyrinth.

The recovery was uneventful, with the disappearance of all symptoms referable to the ear and head. The case was one, however, of latent labyrinthine suppuration, since a sufficient length of time had elapsed to permit of considerable compensation, as indicated by the relative weakness of the spontaneous nystagmus and but slight disparity in the after turning nystagmus to the two sides. There was no involvement of the facial nerve at any time either before or after operation.

Case 2.—G. S., male, 46 years of age, reported first on April 8, 1918, referred by Dr. H. C. Chisholm of Huntington, Pa. History: The trouble began a few months ago with a running left ear following an attack of grippe. The last few weeks he has had severe attacks of vertigo. With the vertigo the patient vomited and was incapacitated. He complains of the hearing in the left ear being poor; never had any trouble with the right ear.

Functional hearing tests reveal normal hearing on the right side and the classical findings of complete deafness on the left, confirmed by the combined speaking tube-noise producer test.

Otoscopic examination of the left ear reveals profuse creamy colored, tenacious secretion in the canal, which is occasionally blood tinged, according to his family physician. After cleansing the canal the membrane appears to be bulging in the posterior half, with a small perforation, through which protrudes a

firm polyp. Right ear reveals an intact, normal appearing membrane.

Spontaneous nystagmus: Mixed rotary-horizontal nystagmus to the right side when the patient looks to the right, straight ahead or to the left, socalled third degree type. Compression and aspiration nystagmus is negative. After turning nystagmus: horizontal to the left side eight seconds and to the right sixteen seconds. Rotary to the left seven seconds, to the right fourteen seconds. Galvanic test presents the classical findings of unilateral left sided labyrinthine destruction with intact eighth nerve. The caloric test is negative on the left side.

The patient was operated upon after the method employed by the writer. He was sent home in good condition, ten days following the operation, with instructions to his family physician as to the further care of the case. The recovery was uneventful and the ear became dry three months after the operation; however, there was slight weakness of the facial muscles on the operated side for a short time.

Case 3.—Miss H. C. C., age 32 years, was first seen by the writer on August 28, 1917, referred by Dr. W. D. Rowland of Asbury Park, N. J. (at present of Boston, Mass.). The patient gives the history of left sided chronic middle ear suppuration, for which she was treated by Dr. Rowland, who diagnosed a cholesteatoma and necrosis of the attic, for which she received appropriate, conservative treatment. The patient had considerable vertigo at times, associated with headaches.

The functional hearing tests reveal complete deafness on the left side to high, low and middle tones. With the noise producer in the right ear and the speaking tube in the left, she was unable to repeat words spoken with a loud voice into the mouth of the tube. The Weber is referred to the right side; Schwabach very short on the left side; normal hearing on the right.

Otoscopic examination: Right drumhead is of normal appearance and behavior. From the left ear there is a discharge of dirty yellow secretion, thin in consistency and of an odor that suggests the presence of a cholesteatoma. There are a few granulation polyps presenting from the attic region.

Spontaneous nystagmus to the right side when looking straight ahead. Horizontal after turning nystagmus to the right, fourteen seconds; to the left, ten. Caloric and fistula tests are negative on the left side. Galvanic reaction is typically that of left sided labyrinthine destruction, requiring seven M. A. stimulation on that side to reverse the direction of the already existing nystagmus.

Microscopic examination of the flaky particles washed out of the left ear along with the secretion reveals cholesterin crystals.

The patient was operated on September 12, 1917, after the method practiced by the writer. She made an uneventful recovery, with the exception of facial paralysis, which has remained up to the time of this writing.

Case 4.—Mr. J. M., age 25 years, was first seen by the writer on February 10, 1918, referred by Dr. W. D. Rowland of Asbury Park, N. J. (at present of Boston, Mass.). The patient gives a history of chronic middle ear suppuration and repeated attacks of severe vertigo, associated with nausea and vomiting.

Otoscopic examination reveals almost total destruction of the left membrane and the presence of granulation polyps springing from the median wall of the tympanic cavity. The examination repeated on February 20, 1918, gives the same results as on the previous visit. The first examination revealed total deafness on the left side to the high, low and middle tones. With the noise producer in the right ear and speaking tube in the left ear, the patient is unable to repeat words spoken with conversational voice into the mouth of the tube. Weber is referred to the right; Schwabach on the left is very short.

Spontaneous nystagmus: There is slight mixed rotary-horizontal nystagmus to the right side when looking straight ahead. After turning nystagmus is markedly reduced toward both sides, especially the right. The caloric test is negative as is also the fistula test.

The patient was operated upon after the method described above and made an uneventful recovery, with the exception of some weakness in the facial muscles on the left side.

Case 5.—Mrs. R. C. Mc., age 40 years, referred on March 4, 1913, by Dr. Raymond Harris, of Philadelphia. The history

is as follows: Four weeks previously the patient began with grippe, a fever and a cold in the head. There was discharge from the nose and a cough. There had been aching all over the body and a fever that kept up for one week. The second week the patient's condition grew worse and she was compelled to go to bed with severe pain in her left cheek, pronounced by Dr. Harris as a "maxillary sinus empyema." For the last forty-eight hours the patient has been complaining of pain in the left ear and for the last twenty-four hours of severe dizziness. The patient further believes that she is completely deaf in the left ear. At no time has there been any discharge from the ear.

The functional hearing tests reveal normal hearing on the right side and loss of hearing on the left. Caloric test on the left side with cold water, over a period of several minutes, failed to produce any reaction. Spontaneous nystagmus is present to the right side, of the mixed horizontal-rotary type. This examination was made at the patient's home, and it was not possible to make the turning and the galvanic tests because there was no available portable apparatus with which the tests could be made.

This case was an emergency one because of the intense headache and other indications suggestive of serous meningitis and was operated upon that same evening. The operation consisted of a radical mastoid, followed by the free drainage of the labyrinth. The facial nerve was not affected. The dizziness continued for several days following the operation. The third day after the operation the patient complained of intense burning and itching of the skin above the left ear, which marked the beginning of what proved later to be erysipelas. This continued to spread, and mixed phylacogen was given with satisfactory results. The wound did well, and in a week's time the erysipelas cleared up. The patient was discharged from the hospital three weeks after the operation. The nystagmus and dizziness continued for one month with decreasing intensity. Three months following the operation the patient was doing well and there was no aural discharge and no impairment of function in the seventh nerve on the operated side.

Case 6.—A. W., 41 years of age, referred by Dr. L. C. Vattier of Philadelphia, was first seen May 1, 1918. The history is briefly that of right sided middle ear discharge of six months' duration. His hearing is "very bad"; complains of dizziness and feels as though he was falling; has headache a good part of the time. There is a tight feeling in the back of the head and "shooting pains" extending from the back of the neck to the top of the head.

Functional hearing tests reveal the typical fork findings of right sided total deafness, while on the left side the hearing is normal for low, middle and high tone forks. The combined noise producer and speaking tube test reveals no hearing to loud conversational voice on the right side.

The otoscopic examination of the right ear reveals the presence of a moderate amount of flocculent secretion. After syringing the ear the drumhead is found to be completely destroyed. A stump of the hammer handle is still present and in contact with the thickened inner wall. In spite of the fact that the patient claims to become dizzy when he pushes his index finger into the right ear, it was not possible at this examination to produce any dizziness by manipulation of this kind.*

Caloric test: After syringing the right ear with water at 65° Fahrenheit for eight minutes, no definite reaction is observable with the head in the horizontal or erect positions. After turning nystagmus: After ten turns to the left there was no nystagmus to the right, and after ten turns to the right horizontal nystagmus to the left twelve seconds. Galvanic test: Impossible to make on the right side because of the discomfort produced by pressure of the electrode.

Patient was operated on May 8th. Usual radical operation followed up by exenteration of the labyrinth. At the operation considerable necrotic bone was revealed. A complicating factor in this case not revealed until some time after the operation was the presence of syphilis.

*This symptom complained of by the patient suggests the presence of a fistula of the osseous labyrinth when the symptom was present, but at the time of this examination its absence would suggest that a loss of function in the labyrinth has occurred between the time the symptom was positive and this visit, when it was negative.

Unfortunately the writer did not think to have a Wassermann test made and therefore missed this factor until the patient thought to bring the matter to his attention.

Five days after the operation, the patient presented the first evidence of facial paralysis on the right side, when he was unable to close his right eye.

His last visit on January 2, 1918, nearly a year following the operation, shows the facial palsy improved but slightly. He still complains of some dizziness and is exceedingly nervous. The operative wound is completely healed.

The writer is unable to give the reason for the dizziness of which the patient complained a year after the operation unless it is due to the effect of a focal infection on the nerve stem. This is purely a speculation and is offered as a possible explanation, since he has seen at least two other cases of focal infection involving the eighth nerve on the side where the labyrinth had been previously destroyed by a suppurative inflammation.

Case 7.—Mrs. J. C., age 38 years, referred by Dr. W. H. Sears, of Huntington, Pa., was first seen by the writer on February 2, 1915, at Huntingdon, Pa. The history is as follows: Scarlet fever at five years of age, complicated by middle ear suppuration on the right side; nine years later had an attack of acute mastoiditis; six years later the ear became quiescent for about ten years. For the past eight or nine years there has been a discharge from the ear the greater part of the time. About five months ago she became suddenly dizzy, with nausea and vomiting during the attack; she has had two similar attacks of somewhat less severity since that time. The fourth and last attack, three or four weeks ago, was the most severe of all.

Otoscopic examination of the right ear shows the presence of pus and débris. After cleansing the ear the drum membrane and ossicles are found missing in large part, with necrosis of the attic wall.

Hearing tests reveal normal hearing on the left side; complete deafness on the right. With noise producer in the left ear and speaking tube in the right, the patient was unable to repeat words spoken in a loud conversational voice.

Spontaneous nystagmus to the left side. Douching the right ear with cold water for five minutes with the head erect produces neither a change in the nystagmus nor vertigo.

Patient was operated on the same day. Radical mastoidectomy and exenteration of the labyrinth. The recovery was uneventful and there was no involvement of the muscles supplied by the seventh nerve at any time.

Case 8.—G. L. D., age 25 years, laborer, colored, was first seen by the writer on November 7, 1913, referred by Dr. Charles H. Harvey, the general surgeon in charge, who first saw the patient. The history was that of an injury from a blow on the head behind the right ear with a shovel in the hands of a fellow workman, ten days before. Notwithstanding the injury the patient was able to walk. He complained of a "stopped up" feeling in the right ear, and the ear was bleeding at the time and continued bleeding for forty-eight hours. The blood was thin and watery (blood plus cerebrospinal fluid). He complained also of severe headache and presented no external evidence of injury to the scalp where he had received the blow.

The functional hearing examination reveals the following findings: Weber referred to the left side; Schwabach short on both mastoids, more especially the right; Rinne negative. There is complete deafness to high, low and middle tones on the right side. The double "a_1" forks of Bezold reveal complete deafness on the right side. The fork tests show normal hearing on the left side. With the speaking tube in the right ear and the noise producer in the left, the patient could not repeat words spoken into the mouth of the tube with conversational voice.

There is complete facial paralysis involving all branches (peripheral type). Otoscopic examination of the right ear reveals profuse, thick, purulent, nonoffensive discharge, which appears to be under pressure and pulsating. The upper wall of the external canal sags, leaving nothing but a very narrow horizontal fissure in place of the normal canal. Further details are impossible to make out, owing to the marked swelling of the canal. Spontaneous mixed rotary-horizontal nystagmus to the left is present when looking straight ahead.

The patient's physical condition was too unfavorable to allow of any further testing of the labyrinth, for during the examination he would lose consciousness.

The case was operated upon radically after the Kuester-Bergmann method. Before reaching the antrum a fracture of the bone was discovered and a loose piece removed, exposing other fragments. (Figs. 16 and 17.) When all fragments were removed a goodly portion of the labyrinth was missing. The operation was completed, after the method described by the writer so far as the comminuted fracture of the petrous bone would permit. The suppurative process in the middle ear and mastoid spaces had extended to and involved the inner ear, so that one was compelled to operate more extensively than in a case uncomplicated with a fracture. His delirium and left sided hemiplegia, present before the operation, disappeared promptly following. His recovery was complete. The wound healed over after eight weeks with complete cessation of the aural discharge, but without recovery from the facial paralysis.

Case 9.—J. G. J., male, age 5 months, was first seen April 15, 1910, referred by Dr. George C. Webster of Chester, Pa. The history was that of a healthy birth, the offspring of healthy parents, according to the family physician. When the child was three months old there developed enlargement of the cervical glands on the left side. Some time later there developed a left sided ear discharge following a night of crying, presumably from pain. The discharge continued intermittently and facial palsy soon developed.

The child was operated upon the following day after the simple method. Some necrotic bone was found in the region of the antrum; the tegmen tympani, however, was found to be intact. Following the simple operation the facial palsy appeared to improve. At the time of this first operation several enlarged cervical glands were also removed. The appearance of the glands, together with the caries and necrosis of bone about the antrum, suggested tuberculosis. (Fig. 18.) The improvement following the first operation was only transient, and it became necessary to operate again on July 20th. At this second operation the entire necrotic labyrinth was removed in four or five pieces. The destruction of the

labyrinth was so complete that it was possible to see the internal carotid artery free of any bone covering for a distance of about three-fourths of an inch—in fact, all of that part of the artery which normally courses through the temporal bone. The dura of both the middle and the posterior fossæ were intact but slightly congested; however, showing no signs of bulging. A few cervical glands that had enlarged since the first operation were removed at this second operation. The patient made an uneventful recovery and lived for ten years and died of what was pronounced epidemic cerebrospinal meningitis by Dr. Webster and the pathologist of the Crozer Hospital of Chester, Pa., who examined the cerebrospinal fluid prior to death.

Case 10.—John B., age 10 years, referred by Dr. Thomas H. Carmichael of Germantown, Pa., was first seen by the writer on September 27, 1926, with the history of pneumonia two years ago, since which time he has had a discharging right ear. The mother claims that "he does not hear much of anything with the right ear." He has had considerable vertigo at odd times.

Functional hearing tests reveal normal hearing in the left ear. The right one is completely deaf to the high, low and middle tone forks. With the three meter tube applied to the right ear and the noise producer to the left, the patient is unable to hear conversational voice. With the three meter tube to the left ear and the noise producer to the right the patient repeats whispered words spoken one foot from the mouth of the tube.

Otoscopic examination: The right ear shows a distinct narrowing of the lumen of the canal at the isthmus, which makes it very difficult to see the anatomic details; however, after douching out the secretion and inspissated epidermis it is possible to observe the membrane, which is almost completely destroyed.

Spontaneous nystagmus: To the left when looking straight ahead with an intensity equal to one mm. at the equator of the eyeball, recurring once in every three seconds. After turning nystagmus: horizontal to the right side nine seconds, to the left fifteen seconds. Caloric test: Syringing the right ear with 5,000 cc. of water, 55° Fahrenheit, over a period of

eight minutes produces no change in the character or direction of the existing nystagmus, with the head in different positions (erect, horizontally to the right, or to the left shoulder). All hearing and labyrinthine tests were repeated on September 29th with the same results. On this second visit the fistula test was made and found to be negative.

On October 6th, the mastoid was operated upon radically, followed by an operation on the labyrinth. The patient made an uneventful recovery, with the exception of a moderate grade of facial palsy, which has since improved but little. The wound cavity has, however, healed up completely and the ear became dry after seven weeks.

Case 11.—Mr. E. E., age 35 years, referred by Dr. W. H. Sears of Huntingdon, Pa., was first seen by the writer on March 2, 1915, at the Blair Memorial Hospital, Huntingdon, Pa.

Present illness began ten weeks ago with severe pain in the right ear, which persisted for two days, when he called on a local physician, who treated him for the acute condition with satisfactory results for about three weeks, during which period he had resumed his work. For the last seven weeks he has not been entirely free of pain in the ear and the whole right side of the head. Twenty-four days ago his headaches became intense and he developed fever with rapid pulse. Ten days ago he went through a severe attack of vertigo, when he experienced the sensation of objects moving around about him, but he cannot recall the direction of rotation. Seven days ago he was brought to the hospital on a stretcher. The vertigo has gradually disappeared.

Dr. Sears first saw the patient on the 24th of February, at which time there was some disturbance in his equilibrium and a sensation of confusion in the head. There was also some aphasia. Dr. Sears, a keen observer, examined the patient closely and obtained the following findings: Spontaneous nystagmus to the left side. Caloric reaction of the right ear negative. With a one meter speaking tube applied to the affected ear and the Bárány noise producer to the sound ear, the patient was unable to recognize words spoken loudly into the mouth of the tube.

Otoscopic examination: Made by Dr. Sears, on March 2. Right ear reveals desquamated epithelium, partially closing the canal. When cleaned away it was possible to see some bulging of the superior posterior wall of the bony canal. The drumhead appears reddened, lusterless and opaque. The anatomic details are somewhat obscured by the discoloration and bulging of the drumhead; the left drumhead appears normal.

Operation the same day, after the Kuester-Bergmann radical method, followed up with an exenteration of the labyrinth as described elsewhere. Because of the suspicion of a complicating cerebellar abscess, the cerebellum was exposed and incised freely but no pus was found. Next the temporosphenoidal lobe was exposed and incised, when a large quantity of pus (at least two ounces) was emptied from a brain abscess.

The patient did well for three weeks, when he took a turn for the worse and died. There was no involvement of the facial nerve at any time following the operation.

Case 12.—H. L., male, age 34 years, referred by Dr. W. H. Sears of Huntington, Pa., on October 24, 1917. History and findings by Dr. Sears. Patient had scarlet fever in infancy with otitis media on the left side; the hearing was never very good on that side. The ear has not been discharging since childhood until several years ago, when he had an acute suppuration of the middle ear again, which cleared and remained dry for a year or two. For the last two years the ear has been discharging at intervals. For the last couple of months the discharge has been continuous. There has been no pain in the ear until a few days ago.

Four months ago the patient had an attack of vertigo one night, falling into bed; the next day he was dizzy and he consulted a physician. A month later (three months ago) dizziness began to annoy him again. The dizziness grew less; however, it persisted to some degree for a month, when (two months ago) he had a violent attack of dizziness, vomiting and disturbance of equilibrium. He went to bed, where he was compelled to remain for three weeks. The vomiting was pronounced for two weeks. The vertigo became gradually less, and at the end of three weeks he was able to get out of bed. He staggered to the left when walking. His normal position

in bed was on his left side; during his illness he lay on his back.

With the onset of the attack, a twitching of the corner of the left side of the mouth and the face developed and remained for four or five days. Since getting out of bed, five weeks ago, the dizziness has gradually decreased, until now he notices it only upon turning his head suddenly, and occasionally when walking he feels a tendency to fall toward the left side.

He has had headaches, left frontal more frequently, occipital occasionally, of moderate severity since the onset of the attack, eight weeks ago. This still persists. He gives no evidence of any intracranial nerve involvement. Eyes are negative. Vision equals 20/20 both eyes. Choked disc or neuroretinitis not in evidence. Extraocular muscles normal. Pupils respond normally to light and accommodation. The left pupil is slightly larger than the right; patellar reflexes normal; no ataxia of upper extremities. The fistula test was negative. The caloric test to the left ear, after five minutes of irrigation with water 68° F., gives no response from either the vertical or horizontal canals. With the Bárány noise apparatus in the right ear and a one meter speaking tube in the left ear he was unable to recognize words spoken in a loud voice. The rotation test: After 10 turns to the right in 20 seconds, with the head erect, there follows an after turning horizontal nystagmus of 10 seconds' duration; after 10 turns to the left in 20 seconds, with the head erect, there follows a horizontal after turning nystagmus of 16 seconds' duration.

The diagnosis of destruction of the left labyrinth by a suppurative inflammation seemed justified from the above findings. The writer was called in consultation, and he concurred in the diagnosis and the opinion that radical mastoidectomy and exenteration of the labyrinth should be done. An extensive radical mastoidectomy on the left ear was done; during the operation cholesteatoma was uncovered and a fistula of the external horizontal canal was observed. This was followed up by an exenteration of the labyrinth.

Following the operation the patient reacted well, with the exception that for the ensuing ten days he suffered with some pain of the gasserian type in the first and second branches of the fifth nerve, which was not relieved for any length of time

by opiates or narcotics; otherwise, the patient made an uneventful recovery. There was at no time following the operation any affection of the facial nerve.

OBSERVATIONS IN LABYRINTHINE SUPPURATION.

1. Most of the cases studied by the writer present the history of repeated attacks of vertigo lasting from a few minutes to a half hour. These attacks of vertigo are most likely due to congestion of the inner ear. If they were caused by any changes in the inner ear of a more severe nature, for instance, serous labyrinthitis, the vertiginous attacks would last longer. Furthermore, in those cases presenting such a history, where the opportunity has been offered for close study during an attack, nystagmus was invariably found to exist, directed toward the side of the ear affected; corresponding, therefore, to what the writer has chosen, with others, to designate clinically as an irritative lesion.

These flitting attacks of vertigo from congestion are not to be confused with the severe final attack which accompanies the destructive type of lesion, as found in labyrinthine suppuration, in which the nystagmus is very pronounced and is directed away from the side of the affected ear.

2. In all cases diagnosed as labyrinthine suppuration, the patient presented the two strongest signs of diffuse destruction of the inner ear, to wit: deafness to loud conversational voice spoken through the speaking tube in the affected ear and the noise producer in the unaffected ear; complete loss of function of the semicircular canal apparatus, as indicated by the inability to produce a change in the character or direction of the existing nystagmus with a stream of cold water played into the external canal of the affected ear over a period of eight minutes or more.

3. In practically all cases there was one outstanding symptom besides those generally accepted as characteristic of diffuse inner ear suppuration, and that was headache. The writer first observed headache as a common occurrence in labyrinthine suppuration as far back as 1907, when he was "Zimmerartz" in the Politzer Clinic and later in Alexander's Clinic. He pointed out to his chief, Alexander, the observation of headache in practically all cases of labyrinthine suppuration. From

that time he has continued to observe the symptom and considers it one of importance, especially since it disappears following the operation upon the labyrinth. It may be looked upon more or less as a danger signal indicating an intracranial complication.

4. Although the writer is unable to trace the end result in all cases that he has operated upon, he finds the number of facial paralyses following the labyrinth operation is approximately 50 per cent, which is rather higher than it should be. The fear of its occurrence tends to deter one from operating as often as the operation is indicated. Facial paralysis occurs in spite of precautions taken against injury to the nerve just below the prominence of the external semicircular canal. It is only recently that the writer has come to the conclusion that the injury to the nerve probably occurs most often as the result of chiseling too high over the promontory and thereby damaging it in the region of the geniculate ganglion.

5. The best evidence that one has operated upon a labyrinth already destroyed by a suppurative inflammation is the finding, not only of pus upon opening the inner ear, but the absence of additional vertigo or change in existing nystagmus following the operation. For instance, given a case of doubtful labyrinthine suppuration in the course of middle ear suppuration, with a remnant of function present but not recognized, the operation of draining the labyrinth would, in consequence of the destruction of that remnant, be followed by an exaggeration of the vertigo and the nystagmus to the side opposite the lesion. Furthermore, where the inner ear is drained in the presence of a purulent inflammation, there follows prompt relief of all symptoms, including the headache; where the labyrinth is more or less intact, the patient feels worse.

6. Following the operation upon the labyrinth the results generally are more satisfactory than after the ordinary radical mastoid, in that healing takes place somewhat more promptly and the cessation of the discharge is more certain. In ten of the twelve cases reported there was complete cessation of aural discharge within a reasonable length of time. In case VI the aural discharge was present one year after the operation. However, this patient had syphilis, besides, it was difficult to keep him under control, in that he was going to sev-

eral other physicians at the same time he was supposed to be under the care of the writer. In the case with complicating brain abscess, the patient's death occurred before there was any chance for the wound to heal.

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1724 SPRUCE STREET.

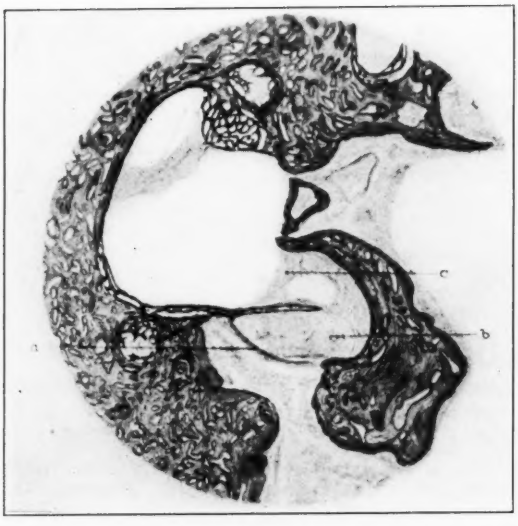

Fig. 1. Taken from Goerke. Labyrinthitis circumscripta. (a) round window; (b) exudate, partly organized in the scala tympani of the basal whorl; (c) band of connective tissue closing off the diseased area from the remaining perilymphatic space. The cysterna perilymphatica is distended by reason of an increase in the perilymphatic fluid. The rest of the labyrinth is intact. This specimen also goes to prove that when both windows are invaded simultaneously the pathologic changes in the labyrinth are more severe in the region of the round than the oval window.

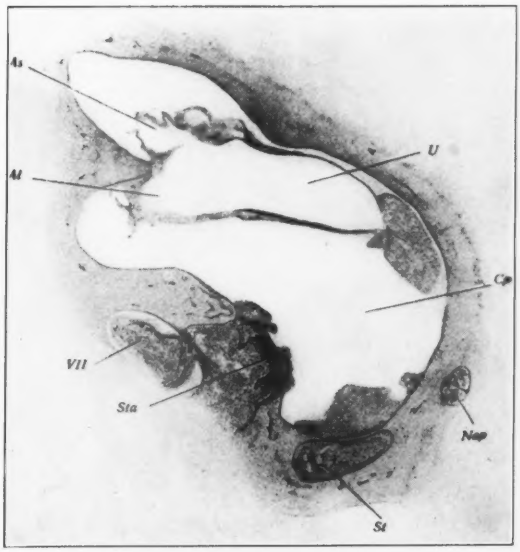

Fig. 2. Taken from Alexander. Adult right ear. Vertical section through the vestibule at the level of the stapes (Sta.). Otitis interna with a collection of pus in the cavum perilymphaticum vestibuli (Cpv), in consequence of an invasion through the oval window with sequestration of the footplate of the stapes (Sta). Utricle (U), superior (As) and external (Al) ampulla remnants. Pus in the scala tympani (St). Small cell infiltration in nervus facialis (VII) and nervus ampullaris posterior (Nap).

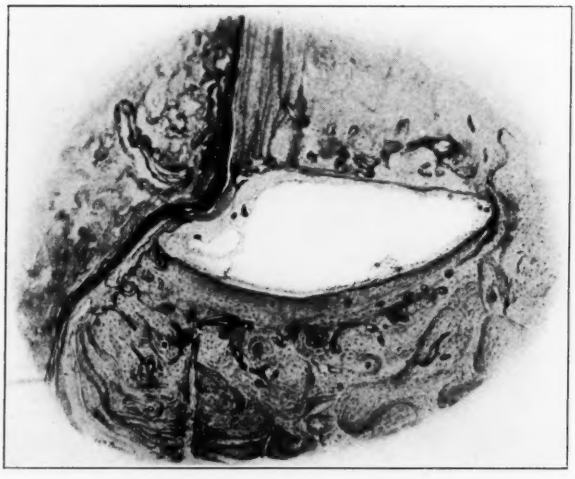

Fig. 3. Is presented by Uffenorde and taken from a specimen in the collection of Professor Wagener. Fistula of the osseous external semicircular canal, developed through the pressure from the growth of a cholesteatoma in the epitympanum. The endosteum is intact. The fistula is uncomplicated by any changes within the lumen of the canal. A case of this kind would probably present no labyrinthine symptoms except through movements, inward and outward, of the exposed endosteal layer produced by compression or aspiration of air in the middle ear spaces (fistula test).

Fig. 4. After Alexander. Chronic middle ear suppuration. Vertical section through the right inner ear of a tuberculous adult and shows (Mts); polyp (Pol) on the lateral surface of the secondary membrane, projecting into the fossula fenestrae cochleæ with secondary atrophy of the vestibular parts of the cochlear canals; (a) atrophied cochlear nerve of the vestibular part; (P) promontory; (St) vestibular end of the scala tympani.

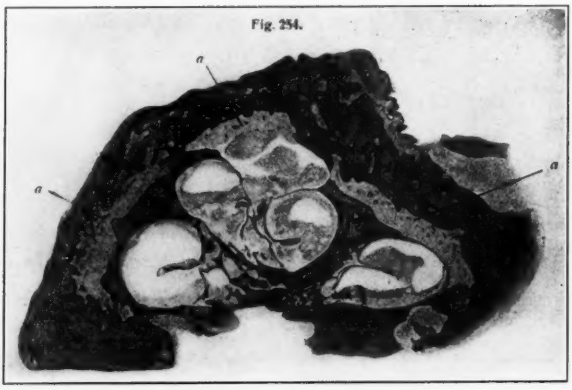

Fig. 5. After Alexander. Vertical section through the right cochlea of an adult, showing chronic suppuration with sequestrum formation (a, a, a) and complete destruction of the soft parts. Remnants of the modiolus are still present.

Fig. 6. Taken from Uffenorde. Acute middle ear suppuration with invasion of the labyrinth through the round window, showing active suppurative inflammation here as well as in the niche of the oval window. A collection of pus cells is seen on the labyrinthine side of the secondary membrane. The case is one of suppurative labyrinthitis followed by meningitis. This case also shows how the labyrinth is invaded through the round window in preference to the oval one.

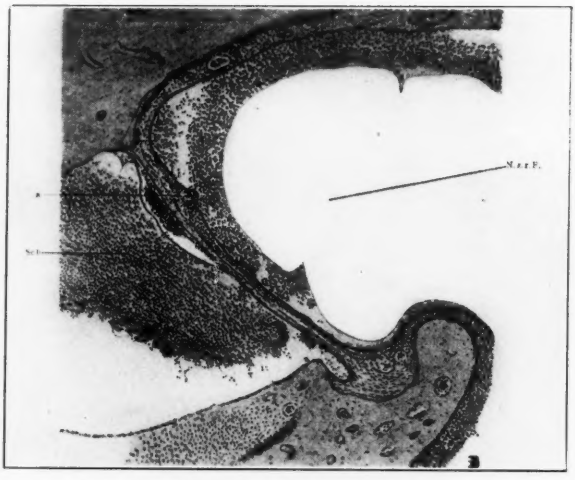

Fig. 7. After Uffenorde. Experimentally produced labyrinth suppuration in an ape. (Streptococcus.) The infection of the labyrinth in this case took place through the round window with the secondary membrane apparently intact. (Window diffusion labyrinthitis.) N. z. r. F. Niche of the round window. Pus with streptococci. The mucous membrane on the tympanic side is intact but infiltrated with pus cells. Sch., Scala tympani of the basal whorl shows many pus cells and some fibrin. X, membrane of round window shows a superficial ulceration on the tympanic side. On the cochlear side, opposite this point, there is a small abscess containing fibrin and pus.

Fig. 8. After Goerke. Healed suppurative labyrinthitis. Region of the round window (a). New formed bone on the osseous spiral process (b). Niche of the round window (c). Organized granulation tissue extending from the mucous membrane lining the niche of the round window, growing into the labyrinthine spaces.

Fig. 9. After Steuer. (Henke and Lubarsch.) Section through a typical semicircular canal erosion showing the fistulous gap in the capsule filled out with connective tissue rich in blood vessels (a). The endosteal layer is still intact. The surface (b) as well as the mucous membrane covering the osseous canal (c) is necrotic. Facial canal (d).

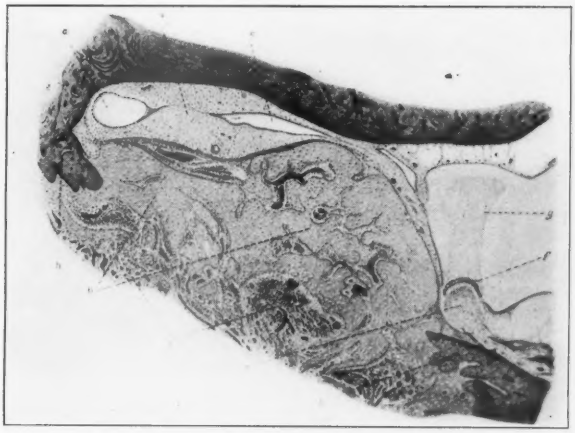

Fig. 10. After Zange. Section through the horizontal semicircular canal after healing of an erosion. New formed hyperplastic bony tissue growing into the lumen of the canal through the original fistula produced by the erosion. Remnants of the original bony resorption tissue (a). New formed bone (b). Narrowed endolymphatic space (c). Perilymphatic space filled with organized connective tissue (d). Well preserved crista (e). Sense epithelial cells (f). Cupola (g). Uninvolved part of labyrinthine capsule (h).

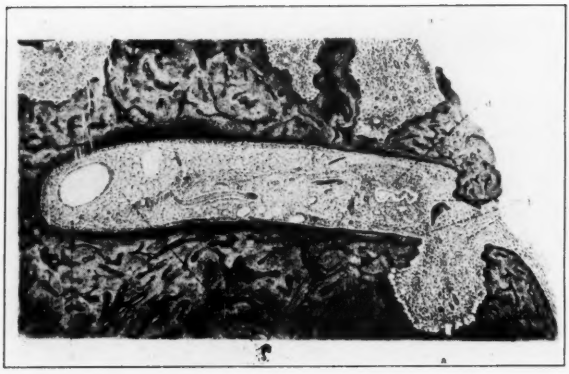

Fig. 11. After Zange. Section through the horizontal semicircular canal of a guinea pig showing erosion of the semicircular canal: Typical bony resorption lesions (a) in which there is contact with the membranous labyrinth (b). Endosteal proliferation leading to organized connective tissue (c). Membranous semicircular canal in cross section (d).

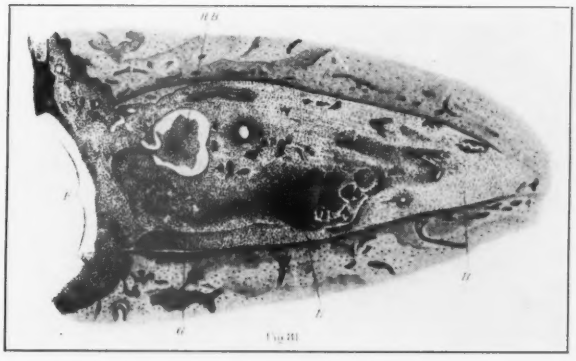

Fig. 12. After Manasse. Oblique section through the horizontal semicircular canal illustrating quite distinctly a fistula formation in the case of a chronic middle ear suppuration. Large fistula in the bony canal (F). The irregular edge of the bony canal is filled out with granulation tissue. The lumen of the canal is filled with pus (E). Granulation tissue (G). Connective tissue (B). The membranous canal is filled with pus (H. B.).

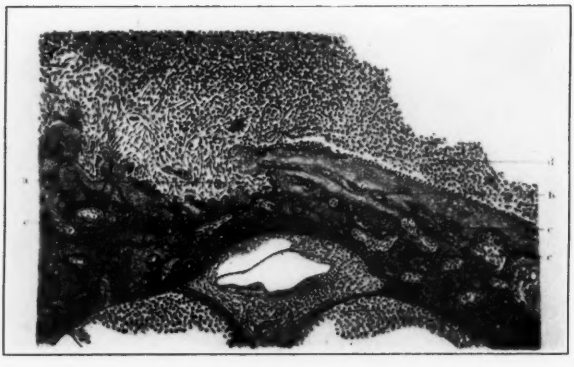

Fig. 13. After Steuer. (Henke and Lubarsch.) Section through a labyrinthine capsule erosion on the promontory. A large erosion on the tympanic side of the promontory (a). Bone of the labyrinth capsule (b). Resorption areas in the process of development (c). Destroyed mucous membrane of the middle ear (d).

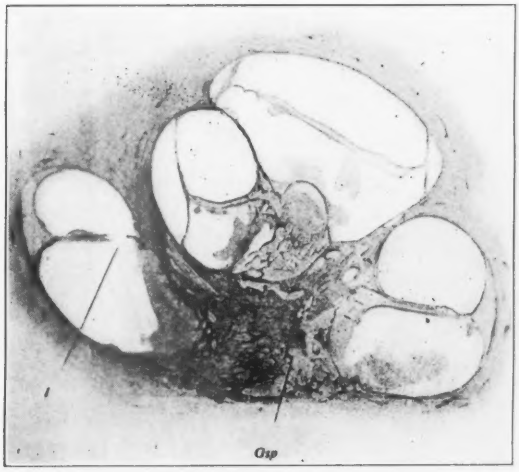

Fig. 14. Taken from Alexander. Axial section through the cochlea showing chronic suppuration of the empyemic type with destruction of the nervous elements. Collection of pus in the scalæ together with circumscribed perforation of the lamina spiralis ossea (L). Ganglion spirale (Gsp) atrophied. A latent suppuration like the one here represented is always a menace to the patient, since any depreciation in the patient's general resistance may be easily followed by the spreading of the infection along the internal auditory canal to the meninges.

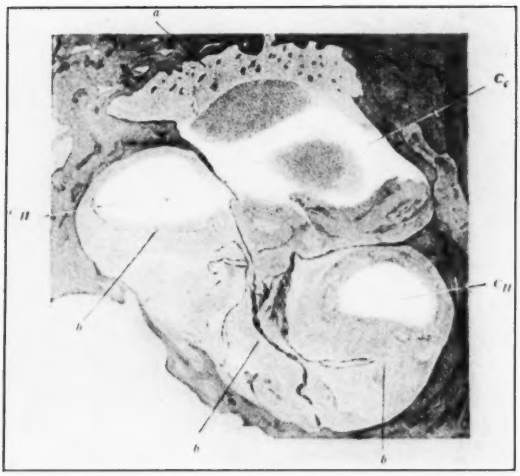

Fig. 15. After Alexander. Cochlea from an adult showing chronic suppuration with sequestration (a) of the capsule. Complete destruction of the soft parts from suppuration (b, b, b). C_{11} middle cochlear whorl; C*c* region about the superior cecum.

Fig. 16. Taken from Alexander. Showing the effects of a fracture of the petrous bone. Cross section through the crus commune of the vertical canals (*Cc*) and the dislocated sinus utricularis superior (Sus). A fragment of broken bone in the perilymphatic space (Fra). Fresh blood and pus in the endolymphatic space (E), and in the perilymphatic space (*Cc*). In a general way this specimen represents in microscopic form the grosser pathologic changes found at operation in Case No. 8.

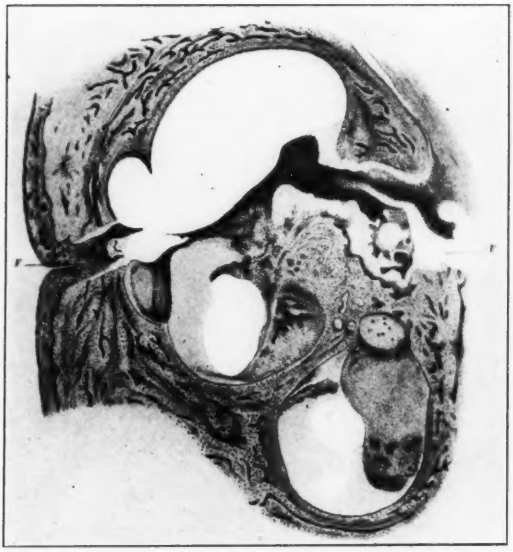

Fig. 17. After Manasse. Otitis interna purulenta acuta traumatica. Horizontal section through the cochlea, showing a fissure (F) that passes almost through the axis. The infiltrated mucous membrane of the middle ear is shown dipping down into the fissure through which communication the suppuration extends from the middle ear to the cochlea. The upper half is empty while the lower half is filled up with pus cells, hyaline and fibrin masses. Most of the soft tissues have been destroyed.

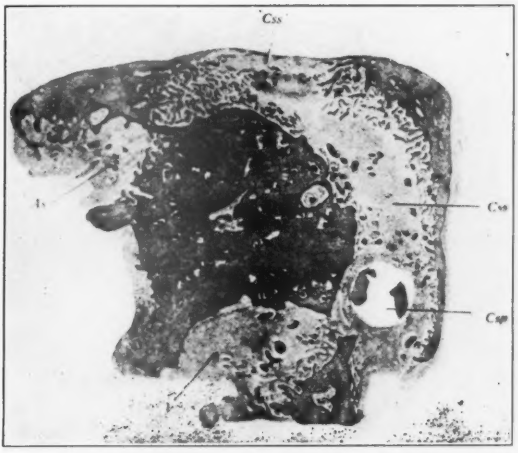

Fig. 18. Taken from Alexander. The illustration is made from a section through the right inner ear of a tuberculous adult and shows caries of the inner ear with complete destruction of the soft parts and bony capsule. The individual parts of the inner ear is rather difficult to make out. The vestibule (V); ampulla superior (As); canalis semicircularis posterior (Csp); canalis semicircularis superior (Css). Other sections through the same bone shows even better the necrosis of the labyrinthine capsule. This illustration is offered as one that probably best typifies, in a microscopic way, the changes found microscopically at the time of operation in Case 9.

XC.

EARLY METASTASIS IN A TUMOR OF THE VOCAL CORDS.

By Arthur E. Hertzler, M. D.,

Halstead, Kansas.

In the December, 1922, number of this Journal, I reported a case in which a malignant lymph gland was removed three years before a growth was first discovered in the larynx, though repeated examinations by competent laryngologists had been made. In the paper above quoted I remarked that "A general surgeon would by instinct have performed an exploratory thyrotomy in order to determine the source of the malignancy when the metastasis in the lymph gland was first discovered."

Recently I was consulted by a patient having a gland identical with that of the patient above noted, operated on five years ago. His history is briefly as follows: Aged 47. His general health has not been good for a year. Six weeks ago he first noticed that he was hoarse and that his throat was sore. At this time he noticed a lump on the right side of his neck on a level with his Adam's apple. It was the size it is now when he first discovered it, and it has remained unchanged since. Examination showed a very hard, somewhat bosselated tumor situated in front of the sternomastoid muscle, at the level of the cricoid ring. It was not freely movable, but there was no definite infiltration of the surrounding tissue. Examination of the larynx showed a tumefaction of the right false cord which covered the true one. The left cord was not affected.

The tumor on the side of the neck was removed by block dissection under local anesthesia. The slide showed definite evidence of malignancy (Fig. 1). I, therefore, in harmony with the resolution above mentioned, did a thyrotomy to find the source of the metastasis and discovered a tumefaction of the right false cord, but without any change in the covering

epithelium. The true cord was not visibly affected, though there was a slight elevation at about its middle. The false as well as the true cord were removed by a small electric cautery.

The slide of the false cord showed an intact epithelium with a considerable cellular exudate beneath (Fig. 2). These cells contained large granular nuclei surrounded by a considerable protoplasm (Fig. 3). These cells resemble very closely those found forming the metastasis in the lymph gland. These cells are obviously not of the epithelial layer. From the study of tumors in other situations (Annals of Surgery, July, 1914, p. 89), I am disposed to regard them as chromatophore cells.

The lesion observed here with the early lymph gland metastasis is so much like lesions in the foot in the paper above cited that I am tendering the hypothesis that it is chromatophore cells which form the tumefaction in the larynx without ulceration and give rise to the early metastasis. This would account for the late appearance of the tumor reported in my case five years ago. This hypothesis would imply a broad view as to the nature of chromatophore cells.

It is, course, a question whether anything has been accomplished by the early removal of the cords in the patient herein reported because the chromatophore tumors situated elsewhere give a universally bad prognosis, no matter how early or radical the treatment. However, if this is the true nature of the tumor, it would account for the unfavorable results from radium in some cases of laryngeal tumor.

These observations may account for the presence of apparently primary epithelial tumors in the lymph glands of the neck in the absence of any primary tumor in the drainage area, which have heretofore been explained on various hypotheses, none of which have received any substantiation.

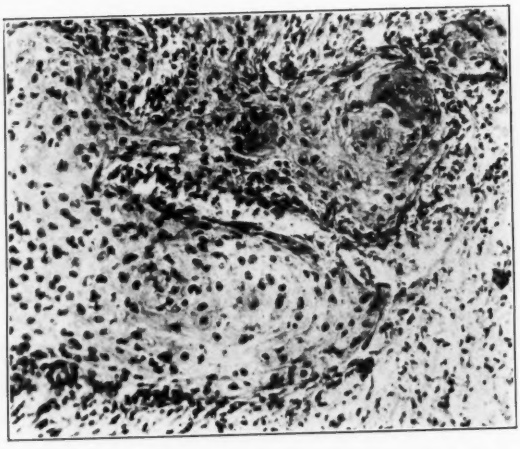

Fig. 1. Epithelium like cell nests in the lymph gland.

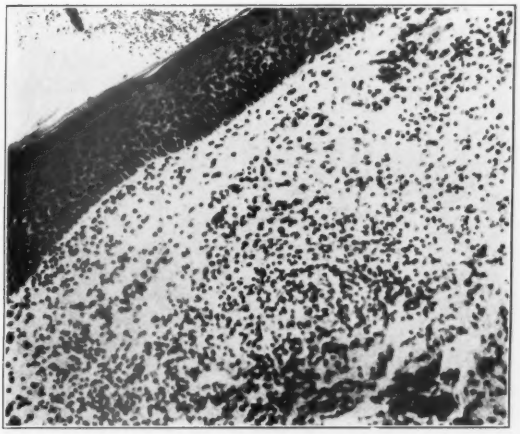

Fig. 2. The mucosa is seen above to be uninvolved. Beneath and unassociated with it are numerous large cells.

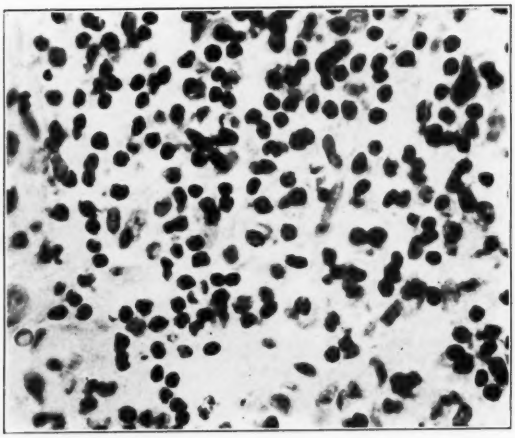

Fig. 3. Higher power of the preceding showing the large granular nuclei.

XCI.

OBSERVATIONS ON THE TREATMENT OF HAY FEVER IN THE PACIFIC NORTHWEST: FIFTY-THREE CASES.

By Charles T. Chamberlain, M. D.,

Portland, Ore.

Although much has been written of late on the various phases of hay fever and allergy in general, very little has appeared in the literature with relation to the hay fever problems of the northern Pacific Coast states.

From a study of the more recent reports, one is impressed with certain facts: First, that greater attention is being given to a more careful and complete study of the hay fever plants and pollen distribution in various localities; second, that there is still considerable variation in the technic employed by different workers in testing and treating their patients; and third, the more intensive treatment is giving an increasing number of successful results.

LOCAL CONDITIONS AFFECTING HAY FEVER.

Hay fever work in Oregon is affected by certain geographic and climatic conditions which make the problem in this vicinity somewhat unique.

Oregon is divided from north to south by the Cascade Mountains. East of the Cascade range is a plateau, with relatively dry climate and high altitude. The winters are moderately severe, and the summers hot and dry with little rain and much wind. Wind-borne pollens of this area are prevalent in the air throughout the entire summer and early fall. The flora is quite varied, including several hay fever plants not found in other parts of the state.

Seasons in the eastern part of the state are fairly regular, and the time of onset of patients' symptoms can be considered a reasonably accurate indication of the group of pollens causing their hay fever.

The majority of all hay fever cases in this locality are caused by the grasses, the Atriplex and Russian thistle, and the Artemisias. The seasons for each are fairly constant, the grasses beginning to pollinate in late May and continuing through June and July, the atriplex and Russian thistle from July to September, and the artemisias from July to October.

In the western part of the state the situation is somewhat different. This area comprises several river valleys, all of which are comparatively low in altitude, with mild winters, as a rule, and abundant rainfall. The flora of these valleys is extremely varied, and it is possible in the space of a few miles' travel to find a complete change in the plants producing hay fever.

The Williamette valley is the largest of these, in which is located the city of Portland. It includes the largest single area of farming land in the western part of the state. Extending north and south and surrounded by mountains on the east, west and south, its climate is characterized by mild winters, summers with few very hot days, fairly frequent rains and little high wind.

The mild winters account for one of the local problems in hay fever work, namely, the great variability of pollinating seasons. The blossoming season of the hay fever plants may be an entire month earlier than the usual date, or after a more severe winter may be delayed for two or three weeks.

Another factor which has to be considered here is that due to frequent rainfall the air is cleared of pollens at various intervals, and patients are often misled into thinking that they have no true hay fever, but only a series of silght "colds." Their family physicians often are at fault in this matter, and therefore many cases do not come for allergic examination until the season is well under way.

The usual hay fever season in the Williamette valley begins with the pollination of the trees of the birch, maple, willow and beech families in late February, March and April. This is followed by the Gramineæ in late May, June and July. Last come the Polygonoceæ, the Chenopodiaceæ, Compositæ, plaintain and amaranth, some of which begin to pollinate as early as May and all pollinating through to September and October.

In the table below are listed some of the most abundant hay fever plants of Oregon, with the approximate dates of pollination:

TABLE A.

COMMON HAY FEVER PLANTS OF OREGON.

WESTERN OREGON (WILLAMETTE VALLEY).

	Pollinating in Early Spring	
*Willow	Salix lasiolepsis	February and March
Hazelnut	Corylus California	March and April
*Alder	Alnus Oregona	March and April
*Birch	Betula alba	March and April
Poplar	Populus	April
*Box Elder	Acer negundo	April
Garry Oak	Quercus Garryanna	April
Ash	Frazinus Oregona	April
	Pollinating During Summer	
Sweet vernal grass	Anthozanthum odoratum	April and May
*Brome grasses	Bromus	May and June
Orchard grass	Dactylis glomerata	May and June
Squirrel-tail grass	Hordeum jabatum	May and June
Tall oat grass	Arrhenatherum elatius	May and June
Velvet grass	Holcus lanatus	May to July
Perennial rye grass	Lolium perenne	May to July
Italian rye grass	Lolium multiflorium	May to July
Johnson grass	Adropogon halepensis	May to July
*Kentucky blue grass	Poa pratensis	May to September
Annual blue grass	Poa annua	May to September
Canada blue grass	Poa compressa	June to July
*Timothy	Phleum pratense	June to July
Fescue grass	Fostuca megalura	June to July
*Couch of quack grass	Agropyron repens	June to September
	Pollinating in Late Summer and Fall	
Black plantain	Plantago lanceolata	May to September
*Sheep sorrel	Rumex acetosella	May to September
Yellow dock	Rumex crispus	May to September
*Lamb quarters	Chenopodium album	June to September
Rough pigweed or red root	Amaranthus retroflexus	June to September

*Also found in Eastern Oregon.

EASTERN OREGON.

	Pollinating During Summer	
Sandberge June grass	Poa Sandbergii	May and June
Crested Koeleria	Koeleria Cristata	June
Wheat bunch grass	Agropyron specatum	
Wheat	Triticum vulgare	
Tall rye grass	Elymus condensatus	
	Pollinating in Late Summer and Early Fall	
Russian thistle	Salsola Kalitragus	June to September
Atriplex	Atriplex hastata	August and September
Sagebrush	Artemisia tripartita	July to October
Mugwort	Artemisia vulgaris	

There has been as yet no complete survey of the state, so far as hay fever plants are concerned. The above lists of plants are the result of several years' experience with hay fever work in this locality, and information furnished by Dr. Helen M. Gilkey of the department of botany of the Oregon Agricultural College.

It is only necessary to glance over these lists of hay fever plants, which comprise only those that are common and occur in relative abundance, to see how futile is the hope of successful results when testing patients with the few test preparations supplied by some commercial house from an extremely limited list of available pollens, several of which are usually of no importance in one's own locality. There are many other plants not included in the above table which have been proved to be the cause of hay fever in isolated cases but which do not occur over widespread areas.

DIAGNOSIS.

The cases which are reported in this paper were studied during the years of 1924 and 1925. They have been divided into two groups because of a change in technic of treatment in 1925. Group I includes cases studied and treated in 1924, and Group II those cases studied and treated in 1925.

These cases were almost all carefully examined for any pathology of the nose and throat, and all such local trouble was remedied before being studied as to allergic reactions.

In studying the allergic condition, careful attention was given to the patient's history, both as to familiar manifestations of allergy and the personal history of complaint, the

time of onset, duration of symptoms, place of residence, effect of any change of residence and occupation. Inquiry was made also as to the ocurrence of any other manifestations of allergic sensitivity, such as asthma, in conjunction with the hay fever, or urticaria and eczema. Not a few patients gave a history of symptoms brought on by contact with other inhalants, especially orris root, house dust and various occupational dusts, and their cutaneous reactions generally bore out the history. Such cases have not been included in this study, which considers only those of uncomplicated pollen allergy.

Testing for reactions to the various pollens was done by the cutaneous method, namely, a linear cut about one-eighth of an inch in length, extending only through the epidermis, on which a drop of the pollen extract was placed. All patients were tested with about fifty different pollens, covering the early tree pollens, the summer grass pollens and the late summer weed pollens.

This was done, regardless of the patient's history as to time of onset and duration of symptoms, first, to show up any slight reactions which might account for a prolongation of symptoms beyond the usual duration, or an earlier onset due to slight sensitivity to some of the early tree pollens; and second, because in some cases we have found patients somewhat indefinite in giving their history as to dates of onset and duration. This is especially true if the case is complicated by reaction to any other inhalants.

Positive reactions to the cutaneous tests were shown as usual by the formation of a wheal, often with pseudopods, surrounded by an area of erythema. A workably accurate idea was obtained as to the degree of sensitivity by the size of the wheal and the presence or absence of pseudopods, especially when the cuts in the skin were made with a sharp knife and with uniform technic. Reactions were recorded as follows:

* = wheal 0.5 cm. in diameter
** = " 1.0 cm. in diameter
*** = " 1.5 cm. in diameter
**** = " 2.0 cm. in diameter

All measurements of the diameter of the wheal were made across the line of the scratch and not parallel to it.

In Table B, the cases studied are classified as to their reactions to the three main groups of pollens and the average strength of reaction in each group. It will be noted that many patients reacted to more than one group having hay fever which extended over much longer than the usual period.

GROUP I

Cases positive to early spring tree pollens			Cases positive to midsummer grass pollens			Cases positive to late summer and fall weed pollens			
Number cases	Per cent	Average reaction	Number cases	Per cent	Average reaction	Number cases	Per cent	Average reaction	Total cases
9	45%	*	19	95%	*** to ****	12	60%	**	20

GROUP II

8	24.2%	* or **	31	93.9%	*** to ****	23	69.7%	** to ***	33

Thus about 95 per cent of all patients tested with pollens in this locality reacted positively to the grass pollens, and reactions to the grasses were usually much stronger than to the other groups of pollens.

TREATMENT.

In the treatment of these cases, all but four were treated preseasonally.

Two different methods were followed, both as to the preparation of pollen extracts and their administration. Cases in Group I were treated with extracts prepared in the manner suggested by Dr. I. Chandler Walker,[1] the 12 per cent alcoholic, normal saline extract, so prepared that 100 cc. of the stock extract represented one gram of the pollen. Sterilization was by Berkefeld filter. Testing of the patients was done with this 1/100 dilution, and various dilutions were prepared for treatment according to the strength of the patient's reactions.

In patients giving strong three plus or four plus reactions, treatment was begun with a 1/5,000 or 1/10,000 dilution. Those giving less positive cutaneous tests were started with a 1/1,000 dilution. The usual doses were 0.2 cc., 0.3 cc., and 0.4 cc. of the 1/5,000, 1/1,000, 1/500 and 1/100 dilutions respectively.

All treatments were carried to a dosage of .5 cc. of 1/100, and this dose was repeated at intervals up to the beginning

of, and in some cases well into the usual hay fever season. Doses were given at intervals of from three or five days. No attention was given to slight local reactions following injection. If reactions became severe, the last dose was repeated once or twice until such reactions failed to appear. Table C shows the results of treatment by this method.

The second group of cases was tested and treated with pollen solutions prepared by extracting the dried pollens in a mixture of 33⅓ per cent saturated sodium chlorid solution and 66⅔ per cent glycerin.

The stock solution in this case was also a 1/100 per cent dilution, 100 cc. representing one gram of the pollen. Sterilization was by Berkefeld filtration. Tests were made with the 1/100 dilution, using the cutaneous method with linear cuts and measuring the diameter of the resulting wheals across the line of the scratch.

Dilutions for treatment were again prepared, according to the strength of the patient's reactions as shown by the cutaneous tests.

The average course of treatment began with the 1/50,000 dilution, then 1/10,000, 1/1,000, 1/500 and 1/100 dilutions. Doses began with 0.1 or 0.2 cc. of the 1/50,000 and then increased rapidly, usually doubling each time, from one dilution to the next lower dilution, until the patient began to show some local reaction to injections. At this point the increase in doses became more gradual, and if reactions became marked, doses were not increased, but the same dose repeated until the reaction failed to develop. Doses were given daily for the first ten or twelve doses and after that every second or third day.

The maximum dose given was 0.2 cc. of the 1/100 dilution, and this dose was repeated up to the beginning of the pollen season in all cases.

In a few cases with strong reactions or with a history of long duration of symptoms weekly injections of the maximum doses were given during at least the first half of the hay fever season. Table C shows the result of treatment in this group of cases also.

RESULTS OF TREATMENT.

Data as to the results of treatment were secured by questionnaires sent to each patient, on which they signified their

own estimate of the amount of relief obtained from their hay fever symptoms.

TABLE C.

GROUP I

Cases Completely Relieved	Per Cent	Partial Relief Cases	Per Cent	Slight Relief Cases	Per Cent	No Relief Cases	Per Cent	Total Cases
8	40%	7	35%	2	10%	3	15%	20

GROUP II

24	72.7%	2	6%	4	12.1%	3	9%	33

A comparison of the results secured from the two different methods of treatment, when administered by the same individual and in several cases to the same patients, may be of more value than if each method were reported by a different worker, from a different locality, working with different pollens, and with probably many other variables in addition to the methods of pollen extraction and treatment.

Some of the details regarding the technic may be mentioned here.

First, all the pollens used in each group of cases were local pollens, collected from the Willamette Valley and near by areas.

The pollens used in Group I were collected by the botany department of Oregon Agricultural College, and the extracts prepared in our own laboratory.

The pollens used in Group II were collected and prepared for use by the Hollister Steir Laboratory.

Second, the study of all cases in both groups, the testing of their pollen reactions, the recording of the strength of those reactions, and the administration of the treatments were all carried out by the same individual, thus assuring a uniform technic throughout.

Third, nine patients are listed in both groups as having been treated by each method, and the comparison of results obtained with them is particularly significant.

Of the twenty cases in Group I, eight, or 40 per cent, reported satisfactory results from their hay fever treatment. Of the thirty-three cases in Group II, twenty-four, or 72.7 per cent, reported satisfactory results from treatment. Satisfactory results have been considered to be either complete relief

of all symptoms of hay fever or relief to such an extent that the patients could go comfortably about their daily work.

It will be noted that there was a much larger proportion of Group I receiving partial relief from symptoms than in Group II, although the total proportion of satisfactory and partly satisfactory results in the two groups is about the same, 75 per cent of Group I and 78.7 per cent of Group II.

Of the nine patients treated with both groups, three received satisfactory results the first year, and there was no appreciable change in results the second year. Five showed a marked improvement in results the second year over the results when treated with Group I. One patient received only slight relief each year.

The above results compare favorably with those reported by other workers in the recent literature: Kahn,[2] 85 per cent perfect results; Spivacke,[3] 76.3 per cent complete or marked relief from symptoms; Piness,[4] 21.4 to 40.1 per cent having from partial to complete relief.

Within the last year much progress has been made in obtaining more satisfactory results by more concentrated treatment given at shorter intervals and in heavier doses, also administering treatment through the entire season at seven to ten day intervals. These changes have produced better general results wherever they have been used.

As to reasons for our failure in some cases, the most important are:

First—The lack of thorough botanical survey of hay fever plants, and, therefore, the possible omission of certain pollens in testing, which may be affecting occasional cases.

Second—Insufficient treatment—dosage not carried high enough, or not continued through the entire pollen season.

Third—The attempt to combine too many pollens in a single treatment solution, especially pollens of different groups of plants.

Of the twelve patients receiving either slight relief or no relief, eight were treated with extracts containing pollens of more than one group of plants, as grasses and weeds, or grasses and trees.

Two of the other four unsuccessful results were in cases treated coseasonally.

The other two coseasonal cases treated with grasses alone received complete relief.

I wish to express my appreciation to my associate, Dr. H. M. Hendershott, for the use of some of his case records, and also to Dr. H. H. Foskett, pathologist to the Emanuel Hospital, for his help in the supervision of all laboratory work.

SUMMARY AND CONCLUSIONS.

1. Approximately 95 per cent of all hay fever in Oregon is of the early or midsummer type, caused by the pollens of the grasses.

2. The cutaneous or scratch method of testing patients for their pollen reactions is satisfactory if performed carefully and with uniform technic.

3. The glycerin saturated sodium chlorid solution method of extracting pollens gives a more potent preparation, both for testing and for treatment, than the 12 per cent alcoholic normal saline extract.

4. More intensive treatment by use of more potent pollen extracts, giving of more frequent doses, and a larger final dose, gives better end results in hay fever treatments.

5. The combining of several pollens, especially if from different groups of plants, in a treatment preparation is not advisable, and may be a factor in failure to secure satisfactory results in some cases.

6. A thorough survey of the botanical flora of a locality must be made, and the hay fever plants, with their distribution and dates of pollination known, before one can hope to obtain the best possible results in hay fever work.

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XCII.

EXPERIMENTAL WORK WITH THE BACILLUS PYOCYANEUS: REPORT OF A CASE OF PYOCYANIC STOMATITIS WITH AGRANULOCYTIC LEUKOPENIA.*

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In 1899, Geoghiewski claimed to have found a leucocyte destroying ferment in cultures of the bacillus pyocyaneus.

In 1924 Lovett reported a fatal case of ulcerative stomatitis associated with extreme leukopenia, in which the bacillus pyocyaneus was associated as the apparent cause. This was the first case described as occurring in this country. During the year prior to this about a dozen cases had been reported in Germany. Since then there have been reported at various times, in this country and abroad, instances of a disease which has been variously known as "agranulocytic angina," "agranulocytosis," or "malignant leukopenia." The total number of such cases described to date is probably between twenty and thirty.

In only two instances in the German series, in Lovett's case in this country and in the two cases to be described below has the bacillus pyocyaneus been found.

The first patient in question was a woman, aged 42 years. Had been married twelve years. Had been delivered of her first child by caesarian section five weeks prior to the onset of her present illness. Pregnancy had been complicated by a pre-eclamptic toxemia. She had been in bed since the delivery of her child, and when first seen for her present illness she was complaining of a sore throat which had developed the previous night. Her temperature was 100°; her pulse 90. There was a small grayish patch on the right tonsil with no involvement of the adjacent lymph glands. The next day the temperature was 101.4°. The right tonsil was clear, but the

*Read before the Pacific Coast Oto-Ophthalmological meeting in Spokane, June 6, 1927.

left one had become involved in a manner similar to the opposite side. The tonsillar condition then apparently cleared up. During the next few days an exudate formed along the gingival margins. It spread to the inner aspect of the cheeks; the floor of the mouth and the ventral surface of the tongue. It did not extend to the tonsils or any other structure outside the buccal cavity. At no time during the course of the disease was there any enlargement of the cervical glands.

The exudate was thick, gray and formed fibrinouslike bands extending from the gums to the inner aspects of the cheeks. It stripped off in large pieces, exposing a surface which did not bleed. The gum tissue between the teeth became necrotic and sloughing. On the fifth day the temperature was 104.° The upper lip became edematous and tremendously swollen, followed shortly by a simliar condition of the lower lip. By the seventh day the temperature was lower, the swelling of the lips was subsiding and the exudate in the mouth was disappearing. On the tenth day she suddenly became worse; pulse poor, dyspneic and very apprehensive. She began to have involuntary stools. She became comatose and died on the eleventh day of her illness.

Autopsy was of little or no help in furnishing additional information as to exactly what type of infection had caused her death. There were ulcerative lesions in the mouth and a few on the genitalia. There was a moderate icterus. The heart was one-third larger than normal and on histologic examination showed acute degenerative changes. The liver and kidneys showed evidence of old and acute infection. There was a healed duodenal ulcer and evidences of previous tuberculosis. There were other findings which had no bearing on the terminal illness, except to indicate that we had been dealing with an individual whose powers of resistance were generally below par. Immediate cause for death was given as an ulcerative stomatitis and acute myocarditis.

A day or so after the patient's death a reexamination of a portion of the membrane obtained from the mouth during the early stages of the disease revealed the presence of numerous bipolar staining organisms which were thought to be those of diphtheria. These were soon found, however, to be actively

motile and gram negative and were finally identified as being the bacillus pyocyaneus. This resemblance to the diphtheria bacillus has been mentioned by other workers, notably by Park in his Textbook on Bacteriology. Gram's method of staining, however, will differentiate these two organisms, since pyocyaneus is gram negative and the diphtheria bacillus is gram positive. Pyocyaneus is easily cultivated in both aerobic and anaerobic cultures and produces a characteristic pigment on suitable media.

Since the original report of a case of this kind in this country in 1924, there have appeared at various times in the literature descriptions of instances of the disease known as agranulocytic angina. The bacillus pyocyaneus in these cases has not been found, except by Lovett.

E. C. Piatt, in a report on the histopathology of agranulocytic angina, discovers in the lesions found in the disease a gram negative bacillus of which he has to say: "If they belong to the bacillus pyocyaneus species, they must possess some peculiarities in their biologic properties." He, furthermore, concludes that the ulcerative areas in the mouth, vagina, spleen, etc., are but "secondary lesions as a consequence of the septicopyemia."

Henry S. Weider reports a case in which the patient suffered two attacks, the second one fatal, and in which the bacillus pyocyaneous was not mentioned.

R. J. Hunter, in 1926, reports a case following a fractured tibia. He reports cultures from the lesions as showing only the usual flora of the mouth.

Harold P. Hill of San Francisco has recently reported a case following the extraction of teeth, in which the bacillus pyocyaneus was sought for and not found, but in which, on account of the blood picture, the diagnosis of agranulocytic angina was made.

Recently we have had occasion to see a case similar to the last one mentioned. Following the extraction of a number of teeth in a middle aged woman, she developed a gangrenous process in the mouth and nose. The total white blood count was 1400, with 3 per cent of granular cells. The blood picture persisted throughout the febrile course of the illness, which lasted two weeks. The white count gradually resumed its nor-

mal proportion and number. Repeated smears from the mouth and nose failed to reveal the presence of the bacillus pyocyaneus. The woman recovered.

On the contrary, Drs. Roy Hammock and Herman Zeiler have recently had occasion to follow a case of ulcerative stomatitis in a middle aged woman, in which the white blood count was 2,400, with 19 per cent of granular cells and in which the bacillus pyocyaneus was recovered in pure culture from the mouth lesions. The woman died.

Clinically this last case and the one described in detail earlier, seem to fall in a definite group, identical with the one reported by Lovett in this country, in 1924, and two found in the German literature. These resemble the cases of agranulocytic anginas described by Piatt, Weider, Hunter, Hill and others, insofar as the blood picture and mouth lesions are concerned, but differ from them in the important details that so far they have always occurred in middle aged women; they are apparently associated with the bacillus pyocyaneus and they invariably terminate fatally.

It would seem, then, that one must distinguish between two manifestations of the same disease. Agranulocytosis, or agranulocytic angina, may occur with any severe stomatitis; it occasionally occurs in men. It has no characteristic bacteriology. There is a leukopenia with diminution in the granular elements of the blood. Such cases are not necessarily fatal. Into this group fall all the socalled agranulocytic anginas.

As a subdivision of this class and one which seems to present more or less of a clinical entity is suggested that group of cases, to date four in number, which apparently occur only in middle aged women, which, in the cases so far reported, have all proved fatal, and in which the bacillus pyocyaneus has been recovered in pure culture from the typical lesions or from the internal organs. For this particular picture is suggested the name "Pyocyanic Stomatitis with Agranulocytic Leukopenia."

Through the courtesy of the clinical laboratory of Drs. Brem, Zeiler and Hammock it has been possible to observe the action of the organism in question on guinea pigs. A culture was made of pyocyaneus obtained from the mouth of the first patient reported, and varying amounts of a broth culture injected into guinea pigs. In order to compare the results obtained,

another organism was chosen, which was known to be either pathogenic or nonpathogenic. For this purpose, B. Coli communis was selected.

From the accompanying charts it will be seen that under certain conditions the bacillus pyocyaneus (or its toxins) is definitely pathogenic for the guinea pig and seems to possess the power of either destroying or inhibiting the formation of the granular elements in the blood, and furthermore that it also has the power to produce a leukopenia; that other organisms, such as the bacillus coli communis, if given in sufficiently large doses, may produce a leukopenia, with the difference, however, that the animal usually recovers.

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7. Note: All laboratory work has been done through the courtesy and in the clinical laboratory of Doctors Brem, Zeiler and Hammock.

No. 1.

CONTROL PIG

	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles	Basophiles	Total count
May 16, 1927						
10:00 A. M.	15	85				13,000
12:00 A. M.	53	44	2	1		
4:00 P. M.	51	46		3		6,900
May 17, 1927						
10:00 A. M.	28	68	2	2		12,700
12:00 A. M.	35	62		3		
2:00 P. M.	32	61		7		7,900
4:00 P. M.	41	51		8		

Control showing variation in differential and total white count.

No. 2.

CONTROL PIG

	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles	Basophiles	Total count
May 16, 1927						
10:00 A. M.	36	63		1		7,100
12:00 A. M.	59	34		7		
2:00 P. M.	66	27		7		18,000
4:00 P. M.	60	35		5		5,200
May 17, 1927						
10:00 A. M.	53	35	3	10		13,900
12:00	33	54		13		
2:00 P. M.	36	48		16		7,400
4:00 P. M.	30	54	1	15		

Control showing variation in differential and total white count.

No. 3.

September 2, 1926

Pig inoculated with ½ agar slant growth Bacillus Pyocyaneus.

	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles
Before inoculation	42	47	1	
2 hours after	79	18	1	2
4 " "	33	66	1	
6 " "	77	18	1	4
22 " "	22	77	1	
26 " "	40	58	1	1
30 " "	22	75	3	
45 " "	12	87	1	
52 " "	11	86	3	

Pig died over week-end.

Pig autopsy I.

Initial leucocytosis followed by moderate leucopenia with relative lymphocytosis.

No. 4.

September 2, 1926

Pig inoculated with ⅕ agar slant growth Bacillus Pyocyaneus.

	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Basophiles
Before inoculation	44	56		
2 hours after	60	40		
4 " "	79	21		
6 " "	91	8	1	
22 " "	68	32		
26 " "	59	40		
30 " "	69	30	1	
45 " "	58	40	2	
52 " "	67	29	4	*
Week later	73	25	2	†

Pig autopsy II.

Sublethal dose showing development of leucocytosis instead of leucopenia.

* Marked leucocytosis. † Slight leucocytosis.

No. 5.

September 13, 1926

Pig inoculated with ½ agar slant growth Bacillus Pyocyaneus.

	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles	Basophiles
Before inoculation	38.5	59.5		2	
2 hours after	53.5	45	0.5	0.5	0.5
4 " "	60.5	37		1	1.5
6 " "	57	39		2	2
24 " "	23	72	3		2
27 " "	17	82	1		†
30 " "	16	82	1		1*
32 " "	17	80	3		*
48 " "	7	87	4		2*
51 " "	8	86	3		3*
56 " "	13	84	3		*
74 " "	1	95			4*
78 " "	4	96			*

Pig died few hours later.

Pig autopsy III.

Almost complete agranulocytosis with moderate dose while organism still virulent.

† Slight leucopenia. * Marked leucopenia.

No. 6.

September 20, 1926

Pig inoculated with ½ agar slant growth Bacillus Pyocyaneus.

	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles	Basophiles
Before inocuation	30	66	1	2	1*
2 hours after	62	37			1†
4 " "	47	52			1†
6 " "	59	38		1	2†

Pig died during night.

Pig autopsy IV.

Animal died before leucopenia had developed.

* Leucocytosis. † Marked leucocytosis.

No. 7.

September 22, 1926

Pig inoculated with ½ agar slant growth Bacillus Pyocyaneus.

	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles	Basophiles
Before inoculation	35	63		2	
2 hours after	69	31			
4 " "	56	42		2	
6 " "	60	40			
8 " "	67	33			
24 " "	6	92	1		1*
27 " "	6	94			*
29 " "	10	90			*

Pig died three hours later.

Pig autopsy V.

Organism recovered in pure culture from throat of animal after subcutaneous injection.

* Marked leucopenia.

No. 8.

November 29, 1926

Tan pig inoculated with 1 agar slant growth Bacillus Pyocyaneus.

	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles	Basophiles
Before inoculation	41	58	0.5	0.5	*
3 hours after	35	64	0	0	1†
6 " "	56	44	0	0	0‡
9 " "	65	33	2	0	0‡
23 " "	28	72	0	0	0§
26 " "	24	75	1	0	0§
30 " "	22	77	1	0	0§

Pig died during night.

Pig autopsy VI.

Culture two months older than when last used—shows decrease in ability to produce agranulocytosis.

* Leucocytosis (?). † Leucocyte ct. lower than before inoculation. ‡ Total count higher than last count. § Leucopenia.

No. 9.

December 13, 1926

Black pig inoculated with 1 slant pyocyaneus.

	Polymorphs	Mononuclears: Lymphocytes	Mononuclears: and Transitionals	Eosinophiles	Basophiles	Total count
Before inoculation	37	49	11	2	1	20,600
4 hours after	66	32	2	0	0	22,700
8 " "	64	35	1	0	0	11,300
24 " "	43	56	1	0	0	7,100
28 " "	21	78	1			5,200
32 " "	22	78	0	0	0	5,000

Pig died during night of December 14.

Relation of differential to total count. Showing leucopenia with relative leucocytosis, using attenuated culture.

No. 1.

September 13, 1926

Pig inoculated with ½ slant B. Coli Communis.

	Polymorphs	Mononuclears: Lymphocytes	Mononuclears: and Transitionals	Eosinophiles	Basophiles
Before inoculation	48	51	1		
2 hours after	75	24	1		
4 " "	61	39	1		
6 " "	67	31.5	1		0.5
24 " "	29	68	3		
27 " "	43	55	2		
30 " "	52	45	2	1	
32 " "	60	39	1		
48 " "	43	53.5	2	1	0.5
51 " "	40	56	4		
56 " "	60	38	1		1
74 " "	32	65		1	1
78 " "	53	45	2		
96 " "	56	40	3	1	
120 " "	30	69	1		
149 " "	59	38	2	1	
173 " "	38	59	2	1	
197 " "	47	52		1	
221 " "	52	48			
245 " "	50	49		1	
269 " "	56	43		1	

Moderate leucocytosis with small dose of B. Coli Communis.

No. 2.

December 13, 1926

White pig inoculated with 1 slant of communis.

	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles	Basophiles	Total count
Before inoculation	49	42	8	1		17,300
4 hours after	79	21	0	0	0	22,200
8 " "	70	29	1	0	0	12,600
24 " "	46	43	1	0	0	19,500
28 " "	44	54	2			14,000
32 " "	62	36	2	0	0	21,600
48 " "	61	37	1	1	0	21,000
53 " "	61	35	3	1		17,400
57 " "	69	29	1	1		17,500
72 " "	49	51	0	0		14,800
80 " "	43	52	1	3		11,500
96 " "	43	53	1	1	2	11,800
120 " "	25	65	5	5		10,200

Pig in good health.

Leucocytosis following larger dose.

No. 3.

September 20, 1926

Pig inoculated with growth 1 agar slant in B. coli Communis.

	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles	Basophiles
Before inoculation	34	61	1	4	
2 hours after	63	35	1	1	*
4 " "	59	35	3	1	2*
6 " "	49	50	1		
24 " "	25	74		1	
28 " "	33	64	2	1	
31 " "	56	39	3	2	
47 " "	31	66	3		
50 " "	37	59	1	3	
53 " "	46	51	3		
71 " "	22	71	3	4	
78 " "	37	59	1	3	
95 " "	20	70	5	4	1
100 " "	41	56	2	1	

No. 4.

September 27, 1926

Pig inoculated with 2 agar slants of B. Coli Communis.

	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles	Basophiles
Before inoculation	26	71		1	2
4 hours after	15	85			
8 " "	57	41	1		1
24 " "	14	83	2		1
28 " "	18	81	1		*
32 " "	21	78	1		*
50 " "	13	83	1		3*
56 " "	15	85			*
73 " "	5	84	11		*
80 " "	5	94	1		*
97 " "	13	87		1	*
122 " "	15	83	1	1	*

* Leukopenia.

Marked leucopenia following large dose; animal recovered.

No. 5.

November 29, 1926

B. Communis.

Brown speckled pig inoculated with whole slant communis.

	Polymorphs	Lymphocytes	Mononuclears and Transitionals	Eosinophiles	Basophiles
Before inoculation	68	29	2.5	0.5	
3 hours after	76	22	1	0	1
6 " "	86	14	0	0	0
9 " "	75	24			1
23 " "	60	37	1	2	
26 " "	46	52	0	1	1*
30 " "	31	65	1	0	3*
48 " "	7	92	1	0	0*
52 " "	10	89	1	0	0*
56 " "	12	79	0	0	9*

Pig died during night of December 1, 1926.

Pig autopsy VII.

* Leucopenia.

Marked leucopenia with death.

SOCIETY PROCEEDINGS.

OPEN MEETING OF THE AMERICAN OTOLOGICAL SOCIETY, HELD IN NEW YORK, MAY 20, 1927.

Address.

By E. B. Dench, M. D.,

New York.

Perhaps no more serious condition can attack those in early life than a progressive impairment of hearing. The failure of the power of audition in early life is frequently not recognized until it has made great advances. Beginning often in childhood as well as in early adult life, this impairment of function advances so insidiously that it is seldom recognized until valuable time has passed and the conditions, even in favorable cases, is well beyond medical science.

Perhaps it would be better for the patients if in the incipient stages the progressive impairment of hearing were more marked, for then the disability would be so noticeable that either the patients themselves or their relatives would insist upon attempting to obtain relief.

Many of the pathologic changes which produce this symptom of progressive impairment of hearing affect at first one ear, then later the other. Nature has endowed the human subject with hearing far in excess of that necessary for the ordinary social and business relations of life. To put it differently, if the individual has one good ear and one very bad, the impairment of hearing is frequently not noticed until the involvement of the good ear becomes rather advanced. Those of us who have made diseases of the ear a life study frequently meet a case where the impairment seems to have begun not more than a few months, or at least a few years, prior to the time when we were consulted. Yet, upon examination, we find in many of these cases the hearing in one ear practically gone and the hearing in the other seriously impaired. This is not

because the patient intended to deceive, or to give wrong facts, but simply because he was misled through the great overplus in hearing of his good normal ear. This normal oversupply of audition, if I may call it so, is undoubtedly responsible for the lack of prompt recognition of early auditory impairment in the majority of cases. I wish to emphasize the fact that this failure to observe unilateral auditory impairment is most common. It is not at all confined to the less intelligent members of the community, but is quite as noticeable in those of the higher walks of life. With superperfect hearing in one ear, almost total loss of function in the opposite ear may exist for many years and be discovered accidentally.

I am not going to attempt, for an audience made up largely of physicians, but also of a goodly number of the laity, to go into any technical description of the various causes which may be responsible for the symptom under discussion. Roughly speaking—and I think it is due the audience that they should know something of what causes this symptom—roughly speaking, I say, progressive impairment of hearing may be due to one or two great causes. In the first place, the disease may start in that portion of the ear ordinarily known as the middle ear or conducting mechanism. It consists simply in the inability of this conducting mechanism to transmit the sounds to the deeper part of the ear; this is known as middle ear deafness, and is amenable to treatment, in a very large percentage of cases. This was made strikingly apparent in the decade 1860—1870 by Wilhelm Meyer of Copenhagen, when he brought forward his work on the effect of the removal of adenoid growths in the nasopharynx as a factor in reducing serious impairment of hearing. At the time of his writing, I believe it is stated that cases of serious impairment of hearing in the young was reduced about 60 per cent over former years. This is ancient history to all present, but, thanks to his beacon light, all of these cases are now cared for. It remains for us to follow his example and aid the other cases.

In another set of cases, it is the belief of many that the disease starts and is always confined to the bony tissues surrounding the end organ of the auditory nerve; that these bone conditions develop from some constitution cause, at present obscure; that they advance steadily, as a rule, although some-

times there may be a period of complete cessation of the pathologic process and the unfortunate condition may not advance.

Thirdly, there are undoubtedly cases where the disease may start in the conducting mechanism—that is, in the middle ear—and then changes either resultant or concomitant may take place in the bony structures. There is also a class of cases in which the auditory impairment is due either to changes in the auditory nerve or in the central areas of the brain, excluding cases of tumor of the auditory nerve and of adjacent brain structures in which the presence of the neoplasm is of primary importance and the aural symptoms simply aid us in clinching the diagnosis. These cases are usually due to either a toxemia of the auditory nerve or to changes in the receptive areas in the brain, coming from either toxemia or, more usually, from vascular changes incident to advancing years. These cases, especially the toxic cases, are well worthy of study. Those due to senility should be promptly recognized, and the patient, unless there be some conducting lesion, should be advised to rely upon general hygienic measures for relief. Youth of the ear cannot be renewed, but good function may be conserved here as elsewhere by proper attention to hygiene.

There are many questions which it is necessary for the medical profession to decide upon, if progressive deafness is to be fairly met. In the first place, it is essential that the research department have at its disposal a large amount of anatomic material—that is, of specimens. To this end your committee has sent out appeals to the various hospitals, requesting that, in each case where a patient comes to the postmortem table, the temporal bone shall be preserved and placed in the hands of the committee for investigation.

You may say at once, why do we wish the temporal bones in cases that have no aural history? The reply is that we are not yet perfectly certain that the bony changes which occur in some of these cases may not be found in patients who have had no previous aural history. In other words, is the change in the bone invariably followed by an impairment of function in the organ, or is the change in the bone merely an accompanying factor? A large number of normal bones must be studied if we are to decide this question. In order to work to the best advantage, it is important that every case entering a

general hospital shall have recorded upon the history whether or not there is any impairment of hearing, and, where there is impairment, there should be a careful test made and the results of the examination should be recorded. The making of a satisfactory record takes but little time, and, properly tabulated, it will be of great advantage. In this way the clinical history can be carefully checked up with the pathologic findings.

Another very important factor is the question of heredity in progressive impairment of hearing. It seems to be a well established fact that given otosclerosis in one or the other parent, the effect upon the hearing of successive generations can be fairly well predicted. By otosclerosis I refer to that class of cases of progressive deafness where the conducting mechanism is normal and the symptoms are caused, or are supposed to be caused, by microscopic changes in the temporal bone. This hereditary factor, of course, immediately brings up the question of how far the physician is justified in advising against marriage in cases where one or the other of the contracting parties is suffering from this particular form of progressive deafness, or where there is a history of this disease in the family. All of us who have had years of experience have been obliged to advise against marriage in young adults suffering from this condition. It is a frightful thing to be obliged to make a decision of this kind. But it is still more frightful to have a union of this kind take place and then be obliged to tell the parents that under no conditoin should they raise a family.

The question of heredity is so serious that the induction of abortion is by many considered a justifiable procedure. The importance of solving this problem so that such a procedure will never be necessary should appeal to everyone. For the last twenty years these changes in the temporal bone known as rarefying osteitis have been held responsible for otosclerosis. Now to what are these changes due? Heredity? Yes. But what was the cause of the first case of this kind? Have environment, diathetic conditions, perverted secretion of the ductless gland, caused these changes?

When we are confronted with these questions, the problem naturally seems much broader and the field of investigation

much wider than one would think when the words "chronic progressive deafness" are used to captionize the condition. An investigation of this kind must of necessity include the examination of a large number of normal temporal bones in order that variance from the normal unattended by symptoms may be classified. Then comes the effect of general diathetic conditions, and general constitutional diseases which may possibly cause changes in the structure of the temporal bone without producing the classical symptom which we have always attributed to these changes. Tests as to variations from normal metabolism are important in conjunction with changes which may take place in temporal bones; also a lack or increase in the calcium content of the blood; the possibility of artificial production of these microscopic temporal bone changes in animals, subjecting them to various diets, endocrine secretions, increase or decrease of calcium in diet and their artificial infection by lues or tuberculosis. All are being borne in mind by those associated in this research, and laboratory experiments on animals are well under way.

This may give you some idea of the gigantic task which faces the committee, and the problems which we are attempting to solve. When it is remembered that the actual inception of our investigation is only about two years old, and when you consider how busy your Research Committee must have been, you may perhaps understand why so little publicity has been given to this Research until the present time. It is due to the public, deeply interested in this question, that they should understand what the committee has been doing for the past two years. Remember that two years ago a certain sum was generously appropriated by the Carnegie Corporation for the study of otosclerosis. The American Otological Society immediately conceived the idea of carrying on the investigation on a rather broader basis. Restricting the Carnegie Corporation grant to the investigation of otosclerosis alone—that is, that form of progressive deafness which is characterized, so far as we know, by certain definite changes in the perilabyrinthine osseous structures, they immediately took steps to raise a fund to be devoted to research which would include all causes of progressive deafness. Hence, the need of so much normal anatomic material as well as specimens from cases giving a his-

tory of progressive impairment of hearing due to any cause whatever.

The first step was to prevent any reduplication of effort, hence the literature of otosclerosis and of progressive deafness is being thoroughly abstracted in order to prevent much loss of time in the repetition of work. A complete index of otosclerosis and of cases of progressive deafness where this was the probable cause was the first work undertaken by the committee. This index is complete to 1926 and is being kept by competent secretaries fully abreast of the time, as current contributions are made. In addition to this, the committee has undertaken the collection, review and translation of all the literature bearing on the subject of otosclerosis, and this work has been completed to 1900. This literature is kept at the Research Bureau of the American Otological Society, Inc., at the New York Academy here, and is available to those who wish to consult it. Within the next year the work of collection, review and translation will be completed, and will be published and offered at cost to those who wish it.

All the hospitals in New York City and Brooklyn have been circularized regarding our investigation, and have been asked to cooperate with us in the furnishing of specimens and clinical histories. The responses to this circular letter are very gratifying, all the hospitals signifying their willingness to aid, and already some specimens have been received, despite the fact that our letter was sent only two months ago. Each hospital will later be given more specific instructions as to the removal and preservation of specimens. The committee has at its disposal enthusiastic young men who are ready to render service either in making histories or in attending to the technic of preparing specimens. This work in New York is, of course, but one step. All the hospitals throughout the country will be similarly urged to cooperate in this research.

At one of our sister institutions in a neighboring city, much work has already been done, thanks to the indefatigable efforts of the head of the otologic department, and to substantial financial aid given by one of our large foundations. With the generosity characteristic of the individual, the cooperation of this investigator with the Research Committee is assured.

Those who are not thoroughly acquainted with the difficulties of the investigation can hardly appreciate how much time must necessarily be spent before actual results can be obtained. Think, for example, of the fact that removing the temporal bone in a postmortem examination must be carried out in such a way that no artefact is caused as a result of faulty manipulation; and all specimens must be decalcified—a process which alone takes about two months. One of the greatest difficulties which has confronted the committee is that we have so few men in this country trained to do this work. For this reason, a certain amount of work is being done abroad, through an arrangement with the committee, by Professor Wittmaack of Hamburg. The chairman of the Otosclerosis Committee, Dr. Pierce, than whom no one is more capable or more enthusiastic on this subject, is responsible for enlisting the services of Dr. Wittmaack.

The League for the Hard of Hearing has approached the question from another standpoint, namely, "The Relief of the Condition When Once Present." Our Research Committee will insist on working side by side with the League for the Hard of Hearing, and there can be no doubt that the Research Committee will be of great aid to the League, on the one hand, and that the League will be able to be of great help to us, on the other.

Address.

By Elmer Ellsworth Brown, Ph. D., LL. D.,

chancellor of new york university,

New York.

It is a saying of the oculists that 90 per cent of our impressions of the external world are those of sight. The saying may be true and at the same time misleading, for there are peculiar poignancies connected with our perceptions of hearing which give to those perceptions an extraordinary value. So it has happened in the history of civilization that the ear has made a fair race with the eye as regards its influence on the development of mankind.

The beginning of history and literature are bound up with oral tradition, recitations, the songs of singers. Then came

written language, and the eye made a dash for first place in the competition. From that time on, the written word consoled the deaf, as the spoken word had long consoled the blind. But writing was for the few, and speech was for the many. When the art of printing appeared, it tended to equalize that difference. In the latter half of the nineteenth century cheaper paper, improved mechanics, popular education, all combined to exalt the printed page and give the eye a tremendous advantage. Men began to say, "The orators are dead, long live the newspaper." But the orators, most of them, refused to die, or when they passed away new orators went on binding with the same old spell. So it was in theaters and churches and presidential campaigns. The ear refused to give up its prerogatives. Then magazines and newspapers added pictures, and once more the eye had forged ahead. The telephone and phonograph answered with a new vogue for hearing. Then motion pictures nearly drove the human voice from the American stage; but the next we knew, the radio had come in with its quiet remark, "Stand by for a few minutes and you will hear——" Now our motion pictures are adding the spoken word and the radios are planning to broadcast pictures of their own, and so again the end is only the beginning.

The strong cards of hearing are talk and music. Talk, first of all. There is not one man in a million who can write as he talks. We have to catch the spoken word, or we fail to get the man—a good part of him, at least. Sometimes the listener gains by being deaf. Oh, yes, no doubt of that. Robert Burdette said long ago that a mustache is sanitary; it keeps unwholesome food from entering the mouth. So a certain amount of deafness is salutary: it keeps unwelcome words from entering the mind. But the most of us would rather decide for ourselves what we will admit, for deafness, as a doorkeeper, has no discrimination.

We want to hear, we want to hear. There are some words, spoken today or long ago, that we want to hear till the last echo of them shall die away; to the end of our days we shall want to hear that "sound of a voice that is still."

And then music. We have just been celebrating the centenary of Beethoven. Who can think of that monarch—he said himself, "I, too, am a king"—without recalling the trage-

dy of his life? Beethoven's deafness was no less a tragedy than Milton's blindness, and it added the supreme bitterness that it struck at the vital center of his art. But Beethoven himself refers to it with especial sadness, because of the limitation it put on his life with men. He wrote in his will: "For me there can be no recreation in the society of my fellows, no refined intercourse, no mutual outpourings. . . . I must live like an exile." When he followed the advice of his physicians and went to live in the country, he said, "My ill fated hearing does not plague me here. . . . Sweet stillness of the woods. . . . Almighty in the forests! I am filled with bliss and happiness in forests!" Yet even there he grieved that he missed the sounds of music that others heard. "What a humiliation when one stood beside me and heard a flute in the distance and I heard nothing, or someone heard the shepherd singing and again I heard nothing."

Just at that time or near it, Wordsworth was in Scotland. How different were his experiences! He carried with him his keen ear for the music of nature and the music of our common life. He was writing of his "Solitary Reaper" and her song:

"No sweeter voice was ever heard
In springtime from the cuckoo bird,
Breaking the silence of the seas
Among the farthest Hebrides.

"I listened till I had my fill:
And, as I mounted up the hill,
The music in my heart I bore
Long after it was heard no more."

Yes, to hear music, and to share in the give and take of human talk, these are among the dearest things in life, and for these sources of intimate happiness we are dependent upon our sense of hearing.

Do you realize the progress that music is making in our American life? I mean, in the life of our people generally. That progress may be measured by the advance in music in our public schools. Lowell Mason began it in the schools of Boston ninety years ago, but for half a century the advance was slow. In the eighties, the movement took a fresh start. In the nineties, a host of supervisors of music in the schools, East and West—Frank Damrosch here in New York had his

part—these supervisors took up the work and added greatly to its momentum. In the early days of this century, high school orchestras became common, and mechanical devices for the reproduction of music came into wide use in the schools. In the years since the great war, the development of chorus singing in the schools, on the one hand, and music memory contests on the other, have brought music home to all classes of our people to an astonishing degree. It is not to be forgotten that this common school development has been made possible by the general advance of musical art in America. The schools are echoing back the achievements of the authentic masters of music. And what is coming from it all is a singing people, a singing America. What may it not mean for our national life, for the spiritual life of the world, for civilization, that our America should be a land of song!

But music is typical of the wide range of human experience in which the emotional refinement of a people is peculiarly dependent upon the sense of hearing, and the approach to which, in the broad and large, is through the common school. On its humbler plane, the tragedy of Beethoven is repeated in the lives of thousands of children, whose hearing is already impaired, and in those of other thousands in whom, unknown to themselves or to their teachers, the seeds of future deafness are already sown.

Our public schools, in the more advanced communities, are taking measures to meet this need. Provision has long been made for those totally deaf. But now other children whose hearing is so defective as to retard their advance in the studies of the schools, receive especial attention, sometimes in classes organized for that particular purpose.

The city of New York has made notable advance in this respect. Health day is here a point of departure for the discovery of pupils who require exceptional attention. In many cases it has been found that children, supposed to be mentally dull, have given that appearance because of imperfect hearing. This condition easily escapes the notice of the grade teacher. It is often concealed by pupils or parents, sometimes intentionally, sometimes unintentionally. Now there is a fair likelihood that it will be discovered, and that possibly in time for permanent betterment.

In all of these efforts the schools have, and must have, the help of the medical profession, particularly of the otologists. But, if I am rightly informed, the profession is itself handicapped by lack of certain knowledge of the causes of deafness, particularly in those progressive forms of which the obscure beginnings go back to the age when children are in school.

In every large concern of human society we sooner or later come up against the need of knowledge, and when we reach the limitations of present knowledge we must go out for more. Without fresh researches we are against a wall. We must beat helplessly against that wall or cut through it and build an arch way into better things. It is so everywhere else; it is so in otology. But American otologists cannot leave this research to Europe altogether, and take the results of European studies as they are handed down to us. We must work with the Europeans and carry our end of the load. Our scientists are fully capable of doing their part. America has material resources that Europe lacks. Much shall be required of him to whom much has been given. If America is to show any proper gratitude for her prosperity, it must be through using her wealth for the betterment of human life. Here is a concrete instance of a universal principle. In the great and intimate field of human interest that depend on the hearing of the human ear, America will use her wealth and the genius of her scientists for the general good of the human race.

Address.

By Henry S. Pritchett, LL. D.,

PRESIDENT OF THE CARNEGIE FOUNDATION,

New York.

A layman hesitates to address himself to a group of highly trained members of a technical profession, and especially does he feels this hesitation when his audience is composed of those who work in a sharply defined field of medical science, such as that which deals with the human ear. Many years ago, when the Carnegie Foundation was engaged in its first report on medical education, a kindly university offered me an honorary degree of doctor of medicine. Had I armed myself with this protection I might consider that I had a true mandate to address your society touching the whole duty of otologists. Under

the circumstances, however, I wish to explain in the beginning that I have no knowledge of medicine.

In all its dealings with the medical profession in the last seventeen years, the Carnegie Foundation has never pretended to a knowledge of the theory, of the science or of the practice of medicine. It has assumed to know something with respect to medical education and medical research. Its studies have been altogether in these fields. I am inclined to think that if a chair could be established in some university on the nature of the medical man, the Foundation might possibly find amongst its staff a properly qualified professor. Tonight I venture to offer a few remarks touching the relation of medical research to medical practice and to the medical practitioner. I may say at the beginning that that relation is essentially the same, whether the medical practitioner labor in one field of medicine or another, whether he call himself a general practitioner, a surgeon or a specialist in some designated field, for all these have their roots in the same fundamental sciences and have a common attitude toward research.

In the experience of the various Carnegie institutions in dealing with research, a few grants of great significance have been made to groups of men working in the field of medicine. As examples of this, I note particularly the grant made years ago at the University of Toronto to the group engaged in the study of insulin, to the group in the study of pyorrhea, and the recent grant made to this society for research in the field of otosclerosis.

Today the name research is one to conjure with. One hears it, not only in the universities and the colleges, but in the industrial establishments and in the technical arts. Scientific research, we are told, is the great agency by which human progress is to be accelerated and human happiness increased.

There is, perhaps, as much mistaken propaganda today in the name of research as in the name of religion, of liberty, or of any other great human field of activity. Our universities are turning out an ever increasing army of doctors of philosophy, whose graduate theses are often listed under the banner of research. And this, notwithstanding the fact that the object of the young man in seeking the degree of doctor of philosophy is generally to qualify for a teaching position in the high

school, the college or the university. For it is well known today that no man is competent to teach in any of our higher institutions unless he is duly stamped with a Ph. D.! Boards of trustees, whether of endowed or tax supported institutions, read with pride the long list of researches emanating from their universities and colleges. In the name of research one finds listed such titles as these, taken at random from the publications of various institutions:

"An Empirical Enquiry Into the God Experience of College Students";

"Advertising Laboratory";

"Pedagogy of the Bible";

"Ice Cream Making";

"Women's Departments in Newspapers and Magazines";

"Research in Clothing";

"Research in Milk Production";

"Plerophory in the Spiritual Experience of John Wesley";

"Advertising Campaigns."

As a matter of fact, the work represented by these and hundreds of other titles does not belong to the field of scientific research in any real sense. They are what the Germans call "arbeiten"—exercises—in which the beginner acquires training in the elementary method of scientific study, and are valuable mainly for the experience and training that they bring to the student. They belong to the process of education rather than of research. When one speaks of scientific research, he means a different order of equipment for the individual and a higher order of intellectual and personal performance.

Research has for its object the advancement of human knowledge. It differs from these elementary studies of the beginner not only in degree but in kind. It is the supreme effort of the human mind to advance the cause of knowledge, and genuine scientific research has at least three distinctive qualities.

In the first place, research must concern itself with a problem of real significance, one worthy of the highest effort. In the second place, it must be prosecuted by men who are familiar with the territory already occupied by science and who start their researches on the basis of present day knowledge. Research in the scientific sense is not the effort of the mere

beginner; it is the adventure of the trained and skillful experimenter and thinker, starting from the vantage ground of territory already won. And, in the third place, true scientific research not only deals with a significant problem, it not only presupposes knowledge and the skill that comes from familiarity with the knowledge of today, but, in addition, those who hope to serve in such a cause must make their venture in the spirit of science—that demands devotion, clear thinking, hard work and absolute sincerity to face the facts. This means an order of intellectual effort, of devotion, of fidelity to truth far above that of the efforts of youthful beginners, however honest and important these may be. A man may not become a disciple of research in a moment; he must grow into it by an apprehension of its significance, by the hard work necessary to master that which is already known, and by devotion that is not only unselfish but clear headed.

All that I have just said pertains to the quality of the problem, to the equipment of the researcher, to the spirit in which he works.

On the other hand, it would be a mistake to assume that only the conspicuous leaders are to be reckoned in the list of those who truly research.

Research today is becoming more and more a matter of cooperation, and for two reasons. In the first place, the great mass of observations turned out in all fields of investigation can be dealt with successfully only by the organized efforts of many individuals. It is so much easier to make observations than it is to analyze them and extract from the the truth, that the mass of observations tends always to accumulate in excess of the analyzing energy available in the world of science. The number of observers far exceeds the number of thinkers, and observations tend to accumulate more rapidly than they are digested.

In the second place, the army of observers needs constantly the scrutiny and the sympathy of the great thinkers whose vision reaches back to the past and forward to the future.

Organized research in the world of today is like an army.

In the front line are the great thinkers, who are able to look into the future "as far as human eye can see." Only the few attain to this eminence.

Behind this skirmish line of the great discoverers comes a much larger body, but still working in the true spirit of the investigator. They are not equal to the great and original discoverers, but they perform a service almost equal in value and in significance, for they appreciate and translate into the service of the world the work of these leaders. They verify and extend the work of the great discoverers; they form the first army of occupation in the campaign against ignorance and in the extension of our knowledge of the truth. Their service is not as spectacular as that of a Newton or a Pasteur. They are, nevertheless, animated by the same spirit and breathe the same air. They are the commanders of the regiments and of the companies.

Behind them comes still a third line, also clearly entitled to recognition as genuine men of research. These are the men who have come to a fair mastery of the methods and of the facts in a given field and who apply themselves to observing, testing and developing the details upon which scientific truth must rest. Their work may not involve great originality, but it requires accuracy and a technic sufficient to take up and elucidate a doubtful process, to prove the truth or the falsity of a proposed theory or to secure the observations upon which theoretic knowledge must be founded. This third division forms the main army of research. They are the common soldiers of science.

It is not always possible for the public, or even for the universities, to distinguish clearly between these separate orders of men. Only once in a long while does a thinker of the first rank present himself. It is a great thing to be a scholar. It is a much greater thing to be a thinker. It is fortunate when a country may count among its citizens a few of the first rank of scientific research, but it is equally important that there should be men capable to command companies and men able to carry on the hard and severe work of a private soldier. All three groups render a necessary service in the development of research and all three contribute to the advancement of knowledge.

And this leads me finally to the suggestion to which these somewhat desultory remarks are intended to point. Men who

are busy practitioners of medicine in a special field cannot all be preeminent in the field of research, but it is absolutely true that the practice of medicine, when wholly separated from that spirit and ideal which the service for truth brings, loses the spirit which ought to animate medical science itself. Medicine is at once an art and a science. As long as one must deal with human beings in the practice, he must know how to carry on his work in a fruitful relation to human aspirations and desires and passions. But medicine, in whatsoever branch, ought also to be a true science, and no man can render a full service in the practice of medicine unless he renders also some service in the field of research by which knowledge in his science is to be widened. Very few men in the active practice of medicine can hope to be great leaders in science, but every man in the practice of medicine may take an intelligent and fruitful part in the development of his science.

He who does not obtain this contact, who does not find his work revivified by this spirit, loses that very quality which will make him most useful to the human beings whom he seeks to serve. No better illustration of the interaction between the forces that promote medical art and medical science could be had than the opportunity now afforded to your society for the conduct of a noble piece of research in a field of the highest importance to the health and happiness of human beings. That research cannot be carried on by a few men. Unless the great body of men of this society will interest themselves to cooperate with the committee under whose care the work is being done, unless they will bring to the help of the committee specimens, methods and cases that come under their own observation, and unless they will bring to bear on the solution of the problem not only the methods of the laboratory but also the clinical vision which comes from practice, there will not be present those factors that will make for a victorious solution of the great problem you have undertaken. I venture to say that there is no other service which this group of medical men could render that would, in the long run, be more significant or more helpful than the hearty cooperation of all the members of your society in the important and far reaching research which has been undertaken in your name.

Seeing Sound: The Use and Value of the Osiso in the Scientific Study of Speech and the Accurate Graphing of Speech Vibration.

By Max A. Goldstein, M. D.,
St. Louis.

(ABSTRACT.)

The study of the mechanism of speech dates back to ancient times, but it is only within the last hundred years that attempts have been made to record the waves of motion produced by the voice as speech curves.

In 1856, Leon Scott, of Paris, invented the phone autograph. This consisted of a large funnel shaped mouthpiece leading to a thin elastic membrane equipped with a revolving cylinder coated with smoke. The manometric flame of Rudolf Konig was a modification of this, in which the change of pressure in a lighted gas burner projected against the membrane manifested itself as a change in the size of the flame.

Alexander Graham Bell, inventor of the telephone, an expert in speech and a teacher of the deaf, and Clarence J. Blake, distinguished aurist, of Boston, modified these devices by using the human temporal bone with the tympanum, malleus and incus intact and with a stile of hay substituted for the stapes. This produced very good graphs.

Electricity heralded a new era in the study of speech graphs. Edward Scripture used the phonograph of Edison and the gramophone of Berliner; however, it was impossible to obtain accurate graphs, for many of the delicate and sensitive speech graphs produced by various consonant elements. In 1924, Dr. Irving B. Crandall and C. F. Sacis, of the Research Department of the Bell Telephone Laboratories, described modern methods whereby, with the adaption of the Oscillograph, highly accurate speech waves were secured.

In January, 1927, Mr. Joseph W. Legg, research engineer of the Westinghouse Electric and Manufacturing Company, announced the invention of the Osiso, a portable and modified form of Oscillograph, by means of which the study of the speech elements was so simplified that another important link in the chain of these investigations was forged. On hearing of this invention, in an appeal to Mr. E. M. Herr, president of the Westinghouse Electric and Manufacturing Company, to pro-

vide an Osiso for Central Institute for the Deaf, in St. Louis, I summed up my first impressions of this device as follows:

"I recognize in the practical application of the Osiso many possibilities for speech instruction for the deaf child. He will be shown the wave appearance of a properly made speech element and encouraged to control his voice to produce the same wave. Faulty tone or voice production will be corrected as pupil and teacher see together the actual moving voice picture."

The purpose of my presentation on this occasion is to offer a preliminary report on the research work which has been undertaken at Central Institute for the Deaf with this instrument, which Mr. Legg personaly installed there.

This consists of (1) the development of a new phonetic record of osisograms of all vowel and consonant elements; (2) the preparation of phonetic textbooks for the education of the deaf child by this system; (3) the study of the pathology of speech as recorded by speech graphs in defective phonation; (4) the instruction of the deaf child to correct his own speech, as made possible by the actual visualization of his defective speech graph, simultaneously with the model speech graph of the teacher; (5) the more accurate analysis of qualitative and quantitative phonetics as presented by sensitive osisograms.

Our first procedure has been directed toward the production of a model graph of every vowel and consonant element of speech. For this purpose we have selected a group of trained speech teachers, both male and female, making individual graphs of their several speech elements and comparing the osisograms for quality and intensity. Composite osisograms have also been produced to develop standards.

The practical objective in the use of the osiso will be to familiarize the eye of the deaf child with the model osisograms that represent the elements of sound and speech. These forms should be impressed on the active child brain as readily as the symbols of the alphabet or as the phonetic symbols of shorthand, and it should be possible to teach the phonetics of speech to the deaf child as readily as to teach him reading or writing by the usual symbols of the alphabet. Finally, the visual reception of speech as a rapid motion picture should offer no greater handicap to the deaf child than his normal ability to rapidly read the text of a printed page.

TRANSACTIONS OF THE AMERICAN BRONCHOSCOPIC SOCIETY, ATLANTIC CITY, MAY 21, 1927.

Address of the President.

By D. Crosby Greene, M. D.,

Boston.

Fellow members of the American Bronchoscopic Society: We celebrate this year the tenth anniversary of the founding of this society. In the year 1917 a group of the leaders of our specialty brought this organization into being, under the presidency of Chevalier Jackson. The moving spirits in this undertaking were Hubert Arrowsmith, the first secretary, and the late Henry L. Lynah, to whose enthusiasm and devotion to its objects the society owed its inception and auspicious beginnings. During these ten years the annual meetings have been productive of valuable contributions to the object of this society as described in the second article of our constitution, namely, "The study of diseases and accidents occurring in the respiratory and upper digestive tracts and of borderline conditions and their treatment, medical and surgical, by direct inspection," and have justified its existence amid the plethora of societies devoted to allied subjects.

The success of the meetings has been due primarily to the steady support and contributions of the founders and to their stimulation and encouragement to the younger explorers in the field. It seems to me most appropriate that the first two papers on today's program should be communications from two of them, the leading exponents of peroral endoscopy in this country.

The present flourishing condition of our organization is due in great measure to the activities of one who was missed at the last meeting when he was beginning to recover from a long and critical illness, but who even in his illness did not fail to plan for the affairs and interests of this society of which he is the executive officer. We welcome at his post today our able and tireless secretary, Dr. Henry B. Orton, the

fourth in the succession of able secretaries who have served the society, but second to none in devotion to its interests.

The art of bronchoscopy is one which emphasizes the importance of Osler's axiomatic reply to the question, What are the three most important factors in making a successful physical examination? "The first is light, the second is more light and the third is still more light." The truth which he thus expressed in reference to physical examination in general is found in no department of medicine of greater applicability than in that of otolaryngology, and particularly in that branch of otolaryngology which is represented in this society. The progress of bronchoscopy has, from its beginning in the hands of Killian, depended on the discovery and development of the means, the instruments for bringing light to bear on hitherto invisible regions of the human anatomy, a phase of the subject which was well stressed at our last meeting in Dr. Yankauer's paper on the relative value of proximal and distal sources of illumination. In devising and producing the instruments for dealing with endoscopic problems there is a large and fertile field to engage the inventive talents of the members of such a society as this, and we find that at each annual meeting valuable contributions to the armamentarium and methods of approach to these problems have been presented. The progress in bronchoscopy has not, however, been limited to development along technical lines. We note in the past few years a striking increase of interdependence between our specialty and other departments of clinical medicine, especially those of internal medicine, thoracic surgery and radiology, which has been of mutual advantage. It is unnecessary today to stress the importance of an expert examination of the chest or of an expert X-ray examination, including fluoroscopy, preliminary to peroral endoscopy. We have come to place more and more dependence on the results of these examinations as aids to the speed and accuracy of our own procedures. Especially helpful has been the growing expertness of the roentgenologist, not only in the localization of foreign bodies and lesions in the respiratory tract and esophagus in preliminary diagnosis, but also, at times, in guiding endoscopic procedures at the time of operation. It was in recognition of the value of assistance in these ways, as well as in the interpretation of X-ray pic-

tures, that this society at its last meeting voted, on motion of Dr. Mosher, to invite a few of the leading X-ray men of this country who have been especially identified with our work to participate as members at our meetings. Bronchoscopy is coming to be recognized as an indispensable part of the study and treatment of cases in the domain of the thoracic surgeon in the localization of suppurations and new growths in the lower respiratory tract, the esophagus and in other pathologic states in the thoracic cavity.

It is through cooperation and coordination with these related departments, as well as in improvement in our methods and instruments, that further advance in bronchoscopy is to come.

Findings With the Barium Bougie in Cardiospasm.

By Harris P. Mosher, M. D.,

Boston.

In presenting this paper I run the great risk of opening the floodgates of discussion on a very old subject, namely, is spasm the cause of cardiospasm? I have never denied that there were cases due to spasm, but I have never recognized one. In my cases the X-ray findings and those obtained through the esophagoscope were those of stricture. Long ago, however, I became convinced that in the typical case of cardiospasm the stricture was a soft affair which was easily broken by the pressure of the examining tube, and lately I have held that the stricture was hardly more than an inflammatory gluing of the deep longitudinal folds of the lower part of the esophagus helped out by an accentuation of the normal twist of the esophagus which occurs in this locality. My chief quarrel with the adherents of the spasm theory has been that they cried spasm either without examining the esophagus with the esophagoscope, or by examining it under local anesthesia and with small tubes. It is natural that the esophagus should react to irritation by spasm, as this is the habit of the rest of the alimentary canal. I have felt lonely in not finding spasm and have cut myself off from most excellent company. I have never been thoroughly happy in excluding such a likely cause. Nevertheless, I simply could not prove its presence. To my mind, the adherents of the spasm theory have not proved their

case; often they have been content not to try to. Today I shall present the spasm advocates with what looks like a means of settling in what proportion of cases spasm is still the acting cause, in any given case, and report my first case of cardiospasm. I expect not a little praise for my frankness in this matter and for my generosity.

In order to estimate the ability of an elastic tube like the esophagus to function as a carrier of fluid and food, it is necessary to know its maximum diameter in at least two directions, preferably the transverse and the anteroposterior. The ordinary elastic bougie will give this information, but only at one point, not over an extended area. The barium bag, on the other hand, can be used to map out the esophagus, from the stomach to the clavicle. Since I have been using the barium bougie I have felt that a new chapter in my knowledge of the behavior of the lower end of the esophagus is opening up.

The barium bougie is an elaboration of an old idea, namely, the introduction into the esophagus of a rubber balloon filled with barium and recording by means of the X-ray plate the shape which it assumes.

Of late I have been getting suspicious of my findings with the esophagoscope at the lower end of the esophagus. It is very difficult to be sure of what you see through a twenty-one inch tube, even if you habitually use a large tube, take advantage of the great aid to clear seeing afforded by ballooning and examine the patient under the relaxation of a general anesthetic. For years I have held that an examination at the lower end of the esophagus under local anesthesia and with small tubes amounted to little or nothing. A negative finding so obtained is time thrown away.

I first used the barium bougie in a case of double bend without stricture of the terminal portion of the esophagus. It was reported before this society last year. The bougie was an improvised affair, and was carried through the esophagus and into the stomach by an esophageal string which the patient wore following a gastrostomy. The present bougie is professionally made and is passed by a whalebone staff. It is available, therefore, as one of the first aids to diagnosis in all cases which fall into the cardiospasm group. It has been passed

in six consecutive cases of this nature and saved all but one from an ether examination with the esophagoscope. The end of the bougie has a blunt metal cap, about a centimeter wide, so that the bougie will pass only a relatively large stricture. I have been pleasantly surprised to find how easy it is to pass the bougie and how well the patients tolerate it. There is active retching if the bougie is allowed to rest at the lower end of the esophagus, but if half of the bag is in the stomach and the other half in the esophagus, the correct position for taking the X-ray plate, there is no retching. The bougie is passed by touch and its position checked up with the fluoroscope. Usually it is found that the whole bag is in the stomach and that it has to be drawn up into place, also under the guidance of the fluoroscope.

Two fortunate cases started off the series of six, which I am about to report. The first patient was a carpenter of middle age, who nine years ago was treated in our hospital for some esophageal condition. What this was I was unable to make out. The gist of the matter, however, is that following his treatment he was without symptoms until he came to the hospital, a few months ago, with a bolus of meat lodged in the lower part of his esophagus. The meat was removed under ether anesthesia. The new barium bougie having arrived from the instrument maker meanwhile, it was tried on this patient more as an experiment than anything else. To my delight, it was passed readily by means of the whalebone staff. I was pleased also to find that the balloon, when distended with barium, was readily tolerated. Both the fluoroscope and the X-ray film showed that there was an annular stricture of the esophagus of large caliber about an inch above the dome of the diaphragm. It was at this stricture that the bolus of meat lodged. Below the stricture the esophagus widened out to a transverse diameter of an inch and a quarter, and it entered the stomach fully as wide. I was startled and so were all who saw the plate. Everybody asked, "Can this be the normal diameter of the lower end of the esophagus?" It was about a month before I could answer the question.

The second case, a woman, on whom the barium bougie was used, was a typical case of socalled cardiospasm. She was half

starved and a living skeleton. For six years she had lived on liquids, regurgitating the first half of everything which she drank. Her X-ray and tube findings were typical of obstruction at the terminal portion of the esophagus. After the tube examination she made a miraculous recovery and went out of the hospital eating everything. My diagnosis was a central stricture, which was dilated by the end of the examining tube. Such dramatic cases are the joy of the surgeon doing esophagoscopy.

The carpenter had a mate in leaving footprints on the sands of science and of time. He was not the walrus as of old, but "Bill," the orderly of the private ward of the infirmary. I asked for volunteers for the bag from the staff and the house officers, with no response. Bill, however, came to the rescue. "Bill" is interested in special knowledge because he is a specialist himself, and takes justifiable pride in his specialty and so do the staff. His specialty is giving high enemata with universal success. He took the bag nicely and retained it for more than half an hour without undue discomfort. "Bill" now, to himself and to me, is the uncrowned hero of the hospital.

I give "Bill's" plates at this point, because they establish the normal diameters of the terminal portion of the espophagus. Curiously, the transverse diameter of the esophagus in both the carpenter and Bill measures exactly the same, namely, one and five-sixteenths inches, a little over an inch and a quarter. The antero-posterior diameter of the normal esophagus, as shown by Bill, is practically the same as the transverse, being only an eighth of an inch less. Further, the anteroposterior diameter of the esophagus is only an eighth of an inch less when the diaphragm is contracted, showing that the cura do not appreciably narrow the esophageal opening of the diaphragm when the diaphragm contracts. This is a point on which I have been in doubt for years.

THE FINDINGS IN THE OTHER CASES OF THE SERIES.

The rest of the cases of the series gave the same finding, namely, a tubular narrowing of the terminal portion of the esophagus. The transverse and the anteroposterior diameters of the narrowing were practically the same in a given case.

Measurements show that the tubular narrowing in the series of cases reduced the diameter of the esophagus a quarter to three-quarters of the normal. In one of the cases there seemed to be a ringlike stricture superimposed upon the tubular narrowing.

The tubular narrowing of the terminal portion of the esophagus being the striking and uniform finding in these cases, a number of questions naturally press for answer. Where is the narrowing? In all but one case—Kelley—it was within the cone of the diaphragm and well above the esophageal opening of the diaphragm and so above the crura.

What is the nature of the narrowing? It is probably a fibrosis of the mucous membrane and fibrous tissue layers of the esophagus. The reason for this statement is the fact that in some of the X-ray plates taken on these patients a wave of peristalsis was caught acting in the strictured area. This could not happen if the muscular layer of the esophagus was involved to any great extent.

With a tubular narrowing of the esophagus of such large caliber, why is there esophageal obstruction? Obstruction must be caused by spasm or twist being superimposed upon the stricture. From the evidence which I have been able to accumulate so far, I feel that a tobacco pouch twist is the more probable explanation of the final shutting down than spasm.

What is the cause of the stricture? I believe that the cause of the tubular narrowing to be a previous lower thoracic or upper abdominal infection transferred by contiguity to the esophagus.

WHAT OTHER INFORMATION DOES THE BARIUM BAG GIVE?

The barium bag shows whether or not the lower end of the esophagus is movable or fixed. Normally when the lung tips fill and expand, the lower end of the esophagus is forced toward the middle line of the body half to three-quarters of an inch. The later plate shows that when the lung tips expand, the lower end of the esophagus is pushed forward also one-half to three-quarters of an inch. There is, therefore, a constant movement of the lower end of the esophagus, and the point at which this movement is greatest is within the cone of the diaphragm. It is a double motion, a potential twist, and might readily be converted into a real one.

THE LUNG TIPS.

The anteroposterior plate shows that when the diaphragm descends and the lung tips expand, not only is the tip of the esophagus pushed towards the median line, but the sag of the lower end of the esophagus to the right straightens out. I once felt that the tip of the esophagus moved upward an inch with the ascent of the diaphragm. Recent measurements on plates, however, show that the upward movement is slight, perhaps a quarter of an inch. The bend to the right is due to the fact that when the diaphragm is up and the lung tips deflated the pressure of the inner surfaces of the lungs on the esophagus, from the bifurcation of the trachea downward, is released and the esophagus fills from above and sags to the right. On the other hand, when the diaphragm descends, the median surfaces of the lungs press on the side of the esophagus and it straightens up.

FIBROSIS OF THE CRURA.

For many years I have wondered whether or not in long standing cases of cardiospasm the esophageal opening of the diaphragm, in other words, the edges of the crura as they make this opening, did not actually become fibrosed and the opening slip up and down on the esophagus like the ring of the old fashioned silk purse. Just lately I found at the medical school a baby, dead at term, in which the edges of the crura were actually fibrosed and very much so. In this case, however, the esophageal opening of the diaphragm was much enlarged, the enlargement being due to a hernia into the chest of the upper portion of the stomach, accompanied by an invagination of the greater curvature of the stomach into the herniated upper portion of the stomach. I have never encountered such a case before and it is mainly a curiosity. It does, however, settle the point which I am now discussing, namely, whether or not the edges of the crura ever become fibrosed, proving that they do.

THE CARDIAC SPHINCTER.

There has been much debate about the presence of a cardiac sphincter of the esophagus. Hyrtel described a sphincter but stated that it was incomplete. Hurst, in a recent article in the Lancet, is an exponent of the cardiac sphincter as the cause

of cardiac spasm. He figures a dissection of the esophagus and stomach in a newborn baby and states that the cardiac sphincter surrounds the lower end of the esophagus as a thin muscular band but little thicker than the rest of the esophageal musculature. It extends upward on the esophagus for about an inch and a half, rising a little above the esophageal opening in the diaphragm. He maintains that tonic contraction of the cardiac sphincter, due to failure of an appropriate stimulus from the esophageal nerve plexuses (Auerbach's plexus), is the single cause of esophageal obstruction in cardiospasm. The failure of the nerve plexuses to send the stimulus which should be followed by relaxation of the sphincter he holds to be due to infection and subsequent fibrosis of the nerve ganglia. In three autopsies he has found these pathologic conditions. He makes no mention of the fact that the esophagus is closed momentarily by the expansion of the lung tips every time the diaphragm descends and that this closure occurs in the region where the sphincter is supposed to act and is independent of swallowing.

It is natural that there should be a sphincter at the cardiac end of the esophagus the same as at the pylorus. A few times I have noticed in the X-ray film of the lower end of the esophagus what looked like a cap, suggesting somewhat the familiar pyloric cap.

Frozen sections show that in the cadaver the terminal portion of the esophagus is closed by outside pressure and by twist. On the living you can actually see with the fluoroscope the lung tips close the esophagus. On the dead the cardiac sphincter is not a conspicuous affair like the pyloric sphincter. It is described, as I just said, as only a slight thickening of the musculature. Why should the sphincter be weak in one place and almost overstrong in the other? If you accept the cardiac sphincter as a structure which functions efficiently and are not troubled by anatomic doubts, the whole question of cardiospasm is settled for you, because the treatment was perfected years ago, is efficient and is still the same, namely, dilatation. I cannot as yet get myself into this contented state of mind about the cardiac sphincter. Perhaps the barium bougie may bring me to it.

For many years I have had a growing conviction that the obstruction in the terminal portion of the esophagus, in cases of cardiospasm, was a slight affair. I have repeatedly likened it to a soft stricture, for it readily gives way to the continued pressure of the end of the large examining tube. In the majority of my cases I have found a small central opening at the lower end of the esophagus which would not open on ballooning but which did open in most cases after pressing the end of the examining tube against it for a minute or two. In some of these cases there was also a small crescentic web, generally on the right. I have not been able to demonstrate spasm. On the above findings, I have held that the cases were essentially cases of stricture. In many of the cases, however, the tube and bougie findings gave an unyielding stricture. Many cases of socalled cardiospasm I still believe to be essentially cases of stricture.

The terminal portion of the esophagus in the resting state is nearly if not actually closed by long and deep vertical pleats. There is a striking similarity between this part of the esophagus and the common rubber tobacco pouch, the mouth of which is closed by folds which are so made that they have a permanent spiral twist. The pressure of the smoker's finger tip easily opens the pouch, but when he withdraws his finger the mouth of the pouch automatically closes. I am indebted to Dr. Martin of San Francisco for this very apt similie. I am a bit jealous of him that he thought of it before I did.

Some time ago I made the statement that it seemed as if Nature was trying to get the lower end of the esophagus into trouble. In front is the largest and most unwieldy organ of the body—the liver; behind, the very substantial left crus. Lying on the diaphragm and following its curves and movements are the lungs. The edges of the lower lobes, and especially their anterior median tips lie on top of the crura and follow them down to their attachment to the spine. When the lung tips fill on inspiration, they are seen to momentarily close the esophagus. The left lung tip being larger than the right makes a characteristic impression on the esophagus. Below the point where the lung tips close the esophagus the weak cardiac sphincter comes into play. Furthermore, in this locality the esophagus has a natural bend from right to left,

and the terminal inch and a half is folded on its vertical axis into deep pleats which of themselves almost close its lumen.

A little pathology at the lower end of the esophagus, therefore, is all that is necessary to cause trouble. You can stop the play of a garden hose by giving the hose a kink. You can do the same thing to the esophagus by increasing the natural kink. A thin rubber bag which is not kept inflated or oiled becomes wrinkled, and the wrinkles as they lie together often become glued. The pleated mucous membrane of the lower esophagus does the same thing under the action of slight trauma or infection. This gluing could also be initiated by spasm.

Three of this group of six cases, which showed a tubular narrowing of the lower end of the esophagus, reported on the same morning, a few days ago, for further examination. One (Kelley) had had an ether dilatation with the large esophogoscope and the subsequent passage of a No. 40 elastic bougie and is eating practically normally. The other two patients are much better, the improvement being due, I feel, to the dilatation which accompanied the use of the diagnostic barium bag. Since the first examination of these cases I had gotten the idea that the barium bag, thin as it is, is capable of producing a certain amount of dilatation. The idea also occurred to me that we could dilate these cases under fluoroscopic vision. With the barium bag in the stomach and well filled, it can be drawn into the esophagus and retrograde bouginage under vision accomplished. As the size of the barium filled bag can be controlled, the size of the bougie can be controlled. What I particularly wanted to do in these cases on this particular visit was to try retrograde bouginage in order to see how firm the tubular narrowing of the esophagus actually was—in other words, to see whether I could differentiate between spasm and actual fibrosis.

The first case of the three was the type of patient in which spasm was to be expected—that is, she is exceedingly nervous. For a year she had moderate trouble with swallowing and then developed whooping cough, one of the most spasmodic of diseases. With the onset of her cough, her difficulty in swallowing greatly increased. She is still having a convulsive cough at times. She is well nourished and acts the part of the highly

nervous woman. I was quite proud of my self control and of my powers of persuasion after successfully passing the bag, and more so when I passed it a second time. The second examination showed that the tubular narrowing was the same as at the first examination. It is the longest narrowing and next to the smallest in diameter of all the cases in the series of six. Under retrograde bouginage, under sight and lasting about two minutes, the esophagus was seen to dilate almost to normal width, and the plate snapped with the fluoroscope confirmed this observation. Here, therefore, is a case either of light fibrosis or of spasm. I think that it is probably spasm. If it is, it is the first one which I have been able to prove.

The other two cases were of the nonnervous type. Both were markedly emaciated, one being on the verge of starvation—you have seen her photograph—and both were cases of long standing, the duration in Kelley being six years. Both had a tubular narrowing of large caliber. In neither of these last two cases did retrograde bouginage under sight produce any change in the caliber of the narrowing. My present opinion regarding these two cases is that at the present time they have passed into the stage of tubular stricture.

TREATMENT.

The diagnostic bag is capable of delivering about five pounds of upward pressure. This is relatively slight compared with the pressure of the Plummer hydrostatic bag. The further treatment of cases similar to these last two would be by a stronger bag or by the Plummer bag filled with a solution of barium so that the effect of pressure could be watched. The dark days of esophageal surgery in these cases is rapidly passing.

Holmes of the Massachusetts General Hospital recently made the observation that the inhalation of amyl nitrite would break up peristalsis or spasm of the intestines. He has given the drug to a number of cases of apparent cardiospasm, and the esophagus has opened after its administration. In one of my series of cases the fluoroscope showed marked peristalsis, and amyl nitrite was given. The peristalsis was quieted a little, not abolished, but the lower end of the esophagus did not open. In at least two other cases of this series the patients

had had atropin given without effect before they entered the hospital. The administration of amyl nitrite is a new method of detecting spasm in the esophagus and should be tried in all cases as part of the routine diagnostic procedures.

It has long been known that the esophagus when filled to a certain point with barium—generally to the level of the arch of the aorta—will bougie itself, another proof that the obstruction at the terminal portion of the esophagus is relatively slight. When these cases first present themselves for examination the esophagus is found to be full of fluid. At times the gas bubble of the stomach will bougie the esophagus from below and open it. It is common history to get from a patient that when he eats he will feel the fluid or food stick at the lower part of the esophagus. He then does the natural thing, takes a drink of water and at once gulps up gas, and this is followed by normal swallowing for the rest of the meal.

Recently I hit upon the use of a fizzing Seidlitz powder as a means of emptying the barium filled esophagus. This procedure promises to be of material help. The gas generated distends the esophagus and makes its outline stand out more clearly, besides hurrying the barium, which has halted, into the stomach. Also it gives a large gas bubble in the stomach against which the lower end of the barium filled esophagus stands out more clearly than I have ever seen it stand out before. This bit of new technic is worth trying.

As I now ring down the curtain for a time on this series of cases, stricture still holds the center of the stage (two cases of stricture and one of spasm). I do not know which of these two bad actors will hold it in the next case. I expect to find out by the use of amyl nitrite and the barium bougie.

828 BEACON ST.

Pneumography by Lipiodol: Its Present Uses and Limitations.*

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The principal reason that stimulated us in the production of this paper has been the frequent publications all over the country of socalled "simplified methods" of using lipiodol.

*Roentgenograms by Dr. A. S. Macmillan, Roentgenologist, Massachusetts Eye and Ear Infirmary.

We make the contention that there is no such a thing as a "simplified method" of using iodized oil, if, by its use, we are going to obtain any really helpful information in obscure lung conditions, and we contend that it should only be used in obscure lung conditions when other diagnostic methods have failed. If we are going to be satisfied with the beautiful X-ray pictures of socalled bronchiectasis, which can be produced by almost any method of using the oil, then simplified methods may be used. The term bronchiectasis is very loosely used, and it is surprising to see the difference of opinion among medical authorities about this condition. For example, Lord, in his textbook, states that it is very uncommon and extremely difficult to diagnose. Osler, on the other hand, states that it is usually easy of diagnosis. If Lord is right, then lipiodol is a valuable aid in diagnosing this condition. The very small percentage of lungs showing bronchiectasis postmortem inclines us to the belief that it is uncommon, and that the term is loosely applied to many suppurative lung conditions.

It may be that we are peculiarly situated at the Thoracic Clinic at the Massachusetts General Hospital in the selection of our cases for pneumography. If we are, we consider ourselves fortunate. This clinic was organized a little over a year and a half ago, consisting of an internist and his assistants, a thoracic surgeon and his assistants, a bronchoscopist and his assistants, together with a roentgenologist. All obscure pulmonary conditions, where it has not been possible to make a definite diagnosis, are referred to this group by the various medical and surgical services of the hospital. The patient, therefore, has previously had a thorough study in the wards of the hospital before he comes to the Thoracic Clinic for diagnosis. The question for this clinic to decide is, (1) What is the patient's lesion? (2) Where is the lesion? (3) Is it a medical or surgical case? (4) What treatment is recommended? The entire group meets every Tuesday morning at 9:30 o'clock. The patients, with their complete history, physical findings, X-rays, etc., are brought in, and those unable to leave their beds are visited in the ward. The history and physical findings are first gone over carefully, being read aloud to the group. The X-rays are then examined and discussed, and then the internist and his assistants make an

examination of the patient's lungs. An open discussion of all the findings then goes on among the internist, the thoracic surgeon, the bronchoscopist and the X-ray man. A decision is then reached as to whether bronchoscopy will be helpful in the individual case or not, and the question of lung mapping is left to the bronchoscopist's judgment. The result of this intensive study is that when the patient comes for bronchoscopy the bronchoscopist has all possible knowledge of the case that can be obtained beforehand. No person is bronchoscoped without having first gone through this group.

It is perfectly evident that when cases are so thoroughly studied beforehand, it is more of a problem to add to what is already known about them than it would be if they had not gone through this intensive preliminary study. By this method of selection of cases, we have avoided any such untoward complications and fatalities as were recently reported by Drs. Archibald and Brown[1] on the "Dangers of Introducing Iodized Oil Into the Tracheo-Bronchial System." They report three fatalities from the literature following the use of lipiodol. The fatalities all occurred in tubercular patients or patients having acute lung conditions.

We do not bronchoscope tubercular patients, much less do we put lipiodol into such cases. Neither do we bronchoscope or use lipiodol in acute lung conditions unless it is absolutely necessary.

One of the most difficult things for us has been to be of material aid to the thoracic surgeon in deciding whether the case is favorable for surgery or not. We have tried various methods of using the oil and, as yet, are frank to admit that, in our hands at least, no technic has been developed that makes it of any great use in most lung abscess cases, and we are inclined to agree with Lord[2] when he states: "In our experience lipiodol has not proved of material assistance in the diagnosis of abscess. Abscess cavities usually communicate so imperfectly with the bronchi that the injected substance outlines the bronchi surrounding the abscess, leaving the involved region itself as a blank, and the diagnosis is then made by exclusion."

We have, however, come to the conclusion that no method of using iodized oil is a good one, unless, as a preliminary

step, there is a thorough cleansing of the pus from the tracheobronchial tree.

One method which we used for a time because it seemed the logical one has been found to be misleading. This consisted in thoroughly cleaning out the main bronchus of the affected lung, then locating the terminal bronchus or bronchi from which the pus was seen to exude. This terminal bronchus was then also thoroughly cleaned out by suction, and the heated oil, at body temperature to prevent reflex cough, was then introduced into this terminal bronchus, usually fifteen cc. being used. We thought that in this way we were getting directly at the lesion and eliminating normal lung, but, unfortunately, we found that by doing this we could almost always produce what looked like a lung abscess by simply filling a lobule of lung with the oil and massing it in that region, there being in reality no real breaking down of lung tissue, but rather a superimposing of lipiodol, possibly in dilated bronchi, giving the appearance oftentimes of a large cavity. This area corresponds to what Jackson describes (in his textbook) as follows: "The term 'abscess' is usually loosely applied to the condition of drowned lung in which pus has accumulated in natural passages and in which there is neither a new wall nor a breaking down of normal wall."

Furstenberg and Hickey[3] have also called attention to this massing of the oil producing pulmonary opacities and leading to erroneous interpretations.

It is important to remember that pus exuding from a terminal bronchus does not necessarily mean gross pathology from that area. It may be a reservoir filled by gravity from a pathologic area in the upper or middle lobe.

A patient in which such a picture was obtained by us was operated upon by the thoracic surgeon, and instead of finding an abscess, he found a number of small pockets, which were superimposed and gave the appearance of one large cavity. This case gave us our first clue to the fallacy and led us to experiment on the lungs of dead and live dogs with the oil. The method we use is quoted from Mosher.[4]

"The intact lungs of a dead dog were inflated and kept in this condition. Through a window in the side of the trachea a bronchoscope was introduced, and through this the cannula

for the lipiodol. A terminal bronchus of the right inferior lobe was injected. The oil was carried by gravity also to the left upper lobe. Typical pictures of lung abscesses were obtained in both places. An attempt was made to evacuate the lipiodol from one of the injected areas. Watching the attempt through the fluoroscope showed that this could not be done. Even after the main bronchus to the injected area was slit open, pressure would not clear the lung of the oil. The same experiment was tried on a living dog, minus, of course, actual pressure on the injected area, with the same result.

"These experiments proved that 40 per cent lipiodol injected into the terminal bronchus of a normal lung will give the picture of a lung abscess. In order not to draw false conclusions, therefore, weaker solutions of lipiodol must be used. Experiments are now being carried on to determine what strength of lipiodol must be used in order to avoid a misleading picture. It is felt that the oil, if it is to be of real service, must be introduced with a nebulizer, and experiments along this line have been started."

We have since tried spraying various dilutions of the oil in dogs but found that in order to get it to atomize it had to be diluted so much that it did not show readily in the picture.

Our present method consists of thorough cleansing by bronchoscopy of the bronchi and the terminals. The oil at body temperature is then placed in the main bronchus of the affected lung, about at the spot which best favors flowing by posture to the pathologic area, as determined previously by physical findings and X-rays. The bronchoscope is then withdrawn and the patient placed in the most favorable position for getting the oil into the desired area. So far, by this or any other method, we have not shown lung abscesses, except in rare instances, only by exclusion—that is, by filling a normal lung about the abscess with the oil. We have found it almost impossible to obtain fluid levels of lipiodol in real lung abscesses, and this we believe to be due to the fact that the abscess is usually filled with pus and only draining out its excess of secretion or overflow. Most often the opening through which the abscess is draining is too far removed from the main bronchus to be reached by a bronchoscope.

To date, only two of our lung abscesses communicated directly with a bronchus explorable with the bronchoscope.

Iglauer[5] has also called attention to the difficulty of injecting lung abscess and feels, as we do, that the cavity is filled with secretion or has a very narrow connection with the bronchus.

Clerf[6] states that lung abscesses are rarely seen bronchoscopically, but can be definitely localized by lung mapping. We do not quite understand what is meant by this statement, unless he means that the abscesses are easily localized by exclusion—that is, mapping out the normal lung about them.

Tubercular cases, as we have already stated, were not bronchoscoped, and we have no doubt that the empty cavities in these cases could be easily filled and fluid levels obtained, as claimed by Ballon.[7]

Socalled bronchiectasis can be easily demonstrated. Stenosis or partial stenosis of a bronchus, due to any cause whatsoever, can be demonstrated by the use of the oil, but in these cases we consider that bismuth subcarbonate powder is superior and gives a much better delineation of the stenosed area. We have given the oil by mouth in two cases of suspected tracheoesophageal fistula. In those cases the fistula was demonstrated easily on the X-ray plate and there was no untoward effect from the patient having swallowed the oil.

In conclusion, we wish to state that in our opinion any case which is obscure enough to require pneumography as an aid in diagnosis, is also obscure enough to be entitled to diagnostic bronchoscopy, for the reason that we still believe diagnostic bronchoscopy to be of more importance than pneumography at the present time.

CONCLUSIONS.

1. In our experience simplified methods of using lipiodol are not of real diagnostic aid in obscure lung conditions. Thorough cleansing of bronchial tree as a preliminary we regard as essential.

2. Real lung abscesses can usually be shown only by exclusion—that is, by mapping out the normal lung about them with lipiodol, the reason for this being that they are not empty cavities but are usually filled with secretion and only draining

out their overflow, this outlet being in many cases far removed from a bronchus explorable with the bronchoscope.

3. It is very easy, by injecting a terminal bronchus with lipiodol, to give the appearance of a lung abscess in the X-ray picture.

4. Our best results so far have been by placing the lipiodol in the bronchus of the affected lung at a location favoring its flow by gravity toward the pathologic area, this flow being helped along by change of posture of the patient. But we are of the opinion that a better technic for the use of oil can still be developed.

5. Bismuth subcarbonate powder is superior to lipiodol in certain lung conditions, notably in stenosis of a bronchus, due to any cause whatever, and in foreign bodies.

6. We have found lipiodol swallowed by mouth useful in the diagnosis of tracheobronchial fistula.

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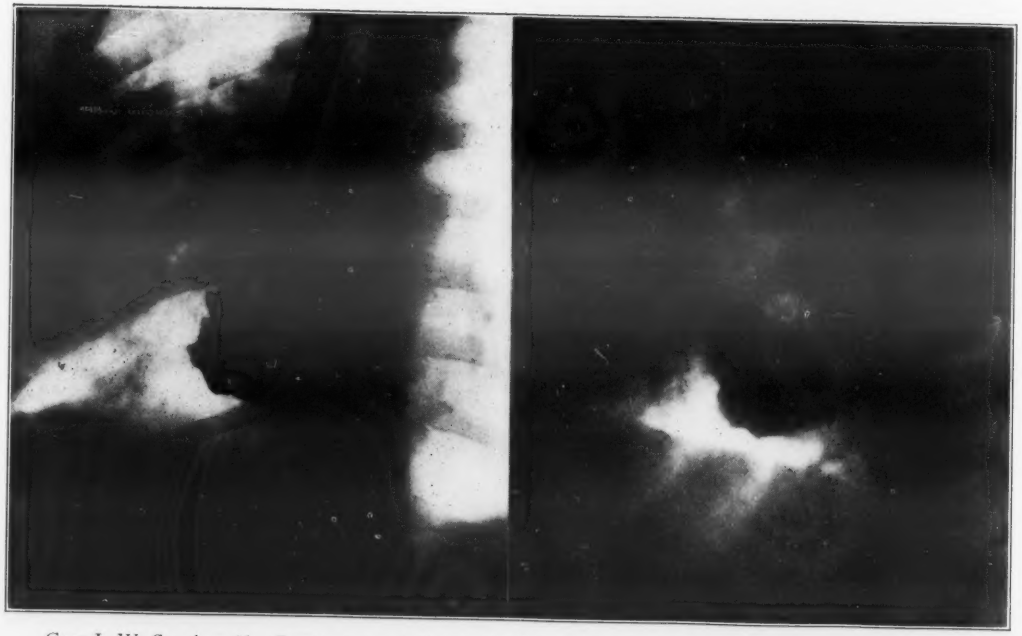
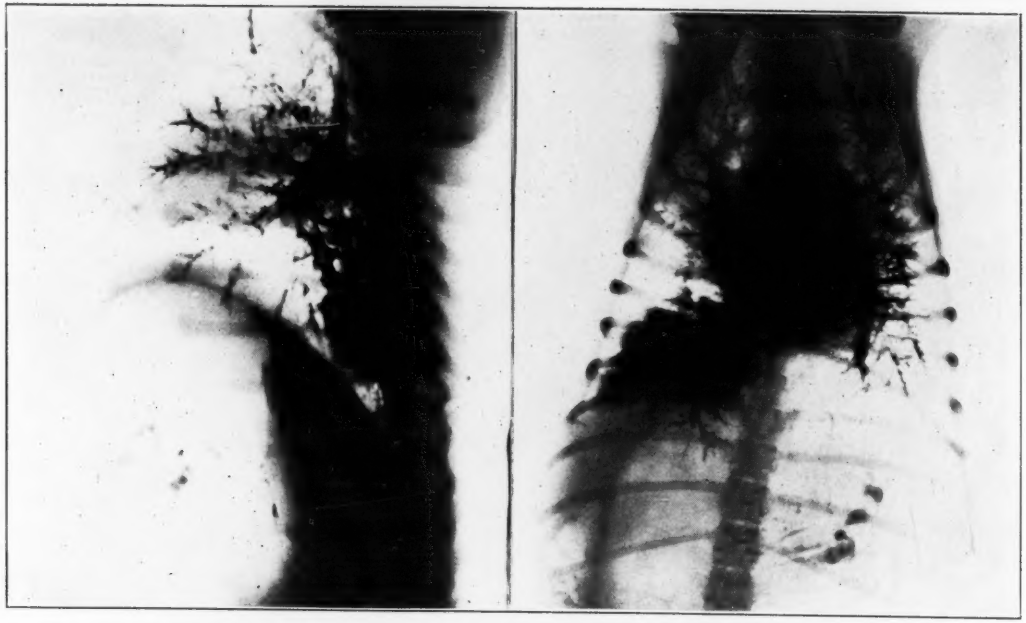


Case I. W. S. Age 44. Symptoms and physical findings were suggestive of a suppurative process at the right hilus. At bronchoscopy purulent secretion was found coming from the posterior terminal bronchus. After cleansing, by suction, 15 cc. of lipiodol was injected for X-ray study. The anterior-posterior view shows a massing of lipiodol in the right lower lobe, surrounded by normal lung. The lateral view shows the mass of lipiodol posteriorly. This location did not correspond to the physical findings, and this case gave us our first clue to the erroneous interpretations due to massing of the lipiodal.

Case H. F. E. L. Age 49. Prebronchoscopic diagnosis, "lung abscess, left lower lobe." Tracheobronchial tree cleansed by suction and lipiodol instilled into the terminal bronchus from which pus was extruding. X-ray studies show a massing of the lipiodol, filling the lobule, producing what has been mistaken for a lung abscess.

To determine whether lipiodol would completely fill a lobule of lung to give an erroneous interpretation, the lungs of a dog were inflated and lipiodol injected through the bronchoscope. This picture of massing of the lipiodol has been mistaken for an abscess.

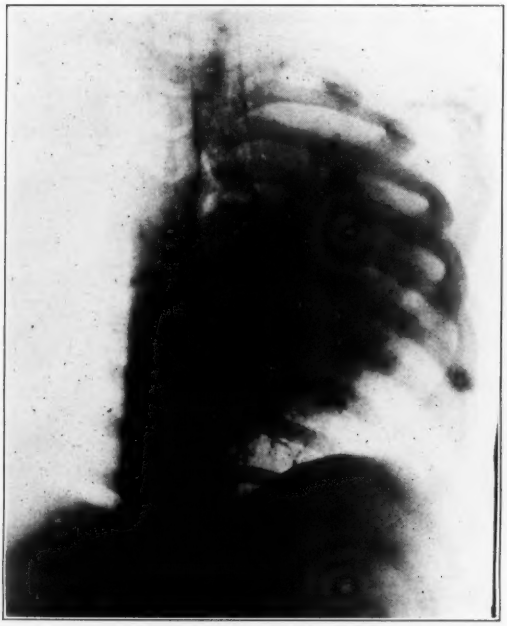


The experiment was likewise tried on living dogs. The lipiodol was injected through the bronchoscope with sufficient pressure only to carry it through the tip of the cannula. By gravity the lipiodol filled the lobule, giving the picture that has been mistaken for an abscess.

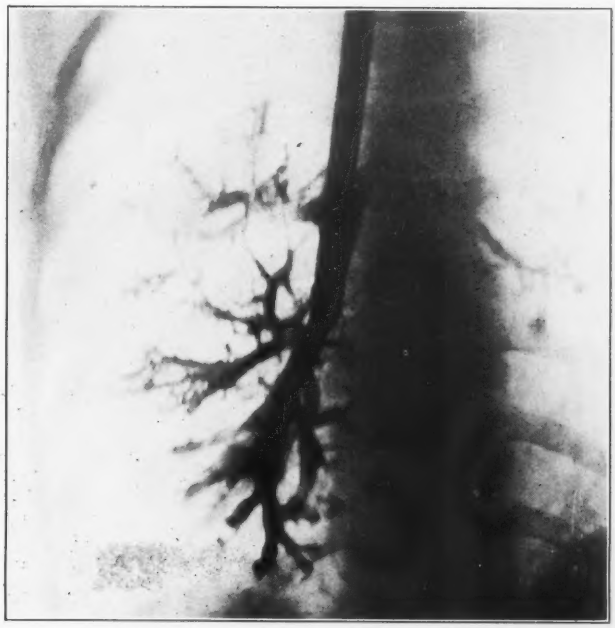
Case III. J. W. Age 38. Symptoms and physical signs of pulmonary abscess of the right upper lobe, following tooth extraction and infection of the lower jaw. Flat plates showed three fluid levels in the right upper lobe. The lipiodol failed to reach the cavities, but pneumography was of value, as the lateral plate shows the lesion to be all anterior.

Case IV. A. K. Age 22. Acute lung abscess following tonsillectomy. This abscess was located posteriorly at the right base, but communicated so imperfectly with the bronchus that bronchoscopic drainage could not be done. The lipiodol, however, did reach the abscess, giving the above picture. This was proven by autopsy to be an abscess.

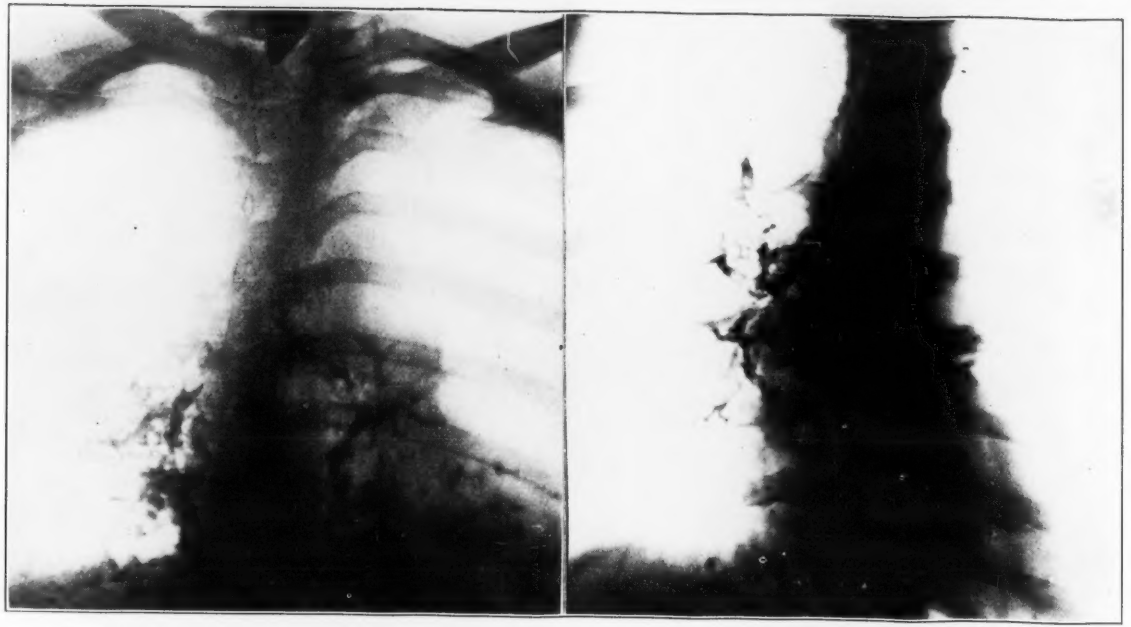
Case V. F. J. Age 29. This case is true abscess. During the bronchoscopy it ruptured, one wall probably being pleura. The patient recovered following rib resection.



Case VI. J. B. Age 16. History of cough with excessive amounts of purulent sputum.
Pneumography by means of lipiodol shows bronchiectasis.



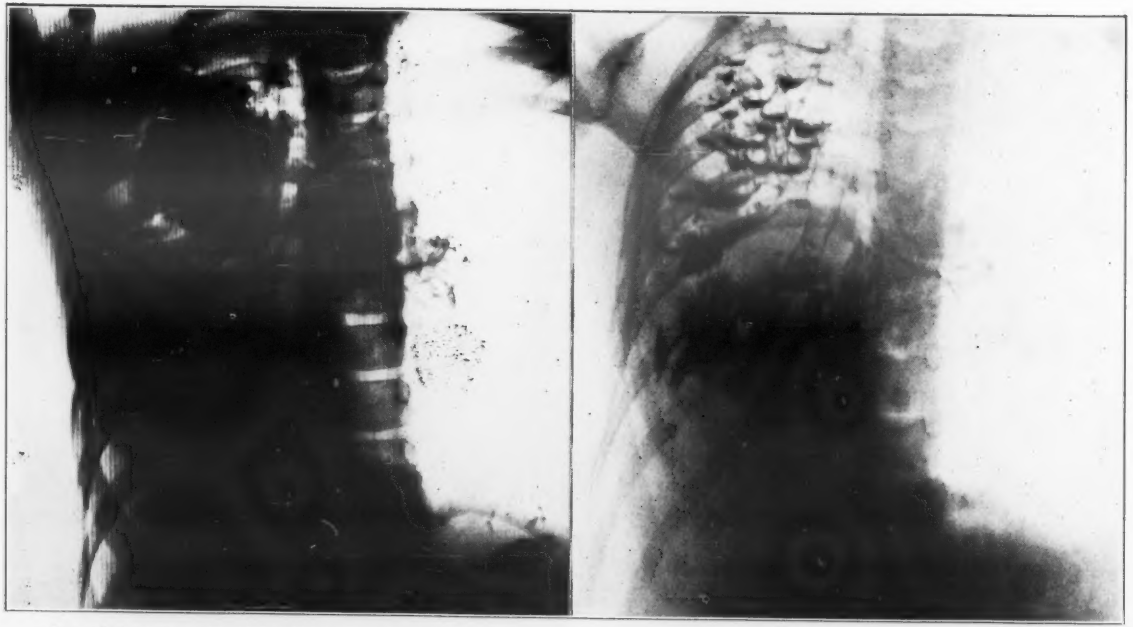
Case VII. C. P. M. Age 16. History of cough with thick, purulent sputum for 4 years. Physical finding suggestive of either bronchiectasis or abscess. Bronchoscopy with pneumography shows bronchiectatic involvement of the right base.



Case VIII. P. M. Age 20. Pneumography shows a unilateral bronchiectasis involving the left base. This patient was considered ideal for an artificial pneumothorax. Complete collapse of the left lung was obtained. The second pneumographic plate is after the collapse of the left lung.

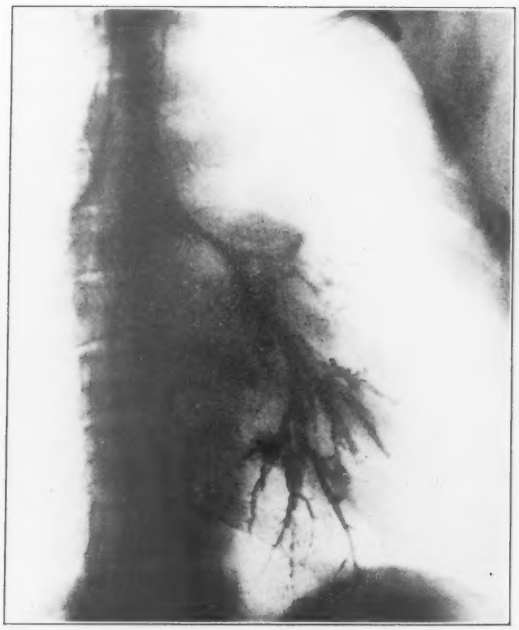
Case IX. M. M. Age 26. History of a lung abscess eleven years ago. In 1917 had a thoracotomy done in two stages with temporary relief. X-ray plates showed no definite cavity, but a diffuse process with some changes in the root region and below it. Bronchoscopy with lipiodol shows bronchiectatic cavities in the right lower lobe.

Case X. D. M. Age 35. Physical signs of a suppurative process of the right upper lobe. The chest plate showed three cavities with fluid levels. Even with the upper lobe curved cannula lipiodol did not reach the abscess cavities, evidently due to blocking of the bronchi.

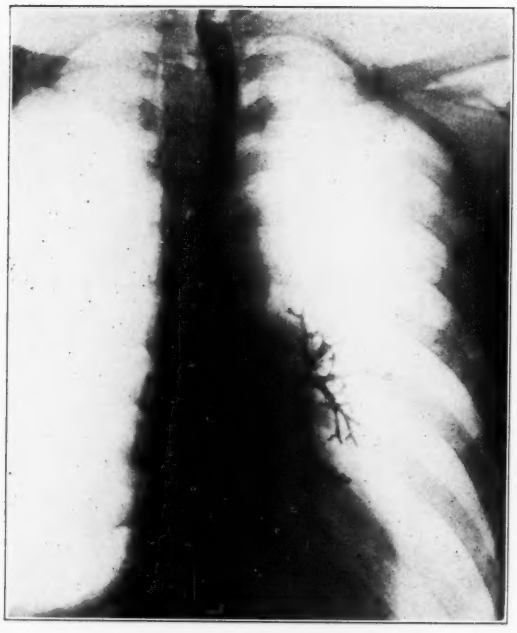


Case XI. T. J. Age 10. History of repeated attacks of pneumonia since infancy, with constant cough and foul, purulent sputum. Physical signs of pathology at the right base. Pneumography shows bronchiectasis of the right base, and what was not suspected, multiple abscess cavities of the apex. Plates taken after coughing show the cavities partially filled, with fluid levels.

Case XII. M. C. Age 46. History of a suppurative process with physical signs of involvement of the upper lobe. Lipiodol injection after bronchoscopic cleansing failed to reach the involved area.



Case XIII. J. S. Age 48. History of cough with excessive amounts of sputum. Physical signs suggested an inflammatory process or neoplasm of the left base. Bronchoscopic examination revealed an incomplete stenosis of the left main stem bronchus. Lipiodol instilled beyond the stenosis and bismuth subcarbonate inflated at the stenosis, shows bronchiectasis of the base, with a stenosis of the left main stem bronchus.



Case XIV. R. F. Age 44. This patient gave a history of difficulty in swallowing semisolid food for the past two years.
Ingestion of lipiodol shows a tracheoesophageal fistula.

Ingested Lipiodol to Demonstrate (1) Nonopaque Foreign Bodies in the Esophagus, and (2) Esophagotracheal Fistula.

By Samuel Iglauer, M. D., F. A. C. S.,

Cincinnati.

Before making a search for a suspected foreign body in the esophagus it is always advisable to employ the X-ray as an aid to diagnosis. A radiopaque foreign body of any size is usually easily demonstrable, but nonopaque bodies present certain difficulties. The usual technic in demonstrating the presence of nonopaque bodies is to have the patient swallow barium in a suspension, or in a capsule, which may become arrested above the intruder. In the subsequent search with the esophagoscope, the barium is frequently seen to form a pool above the foreign body, obscuring the latter. It requires considerable time and effort to remove the barium before the foreign body can be fully exposed to view.

Recently in several cases I have employed iodized oil instead of barium to determine the presence of a nonopaque foreign body in the esophagus, and have found that it has certain advantages which will be enumerated in describing the cases.

Case 1.—Mrs. R., age 44, married, mother of four children, was referred to me by Dr. Starr Ford, March 3, 1927. Her chief complaint was inability to swallow anything.

Previous history: At age of 21 months she accidentally swallowed some home-made lye (wood ash), after which she was very ill for months. All her life she has had difficulty in swallowing. During the past ten years on three or four occasions she has had almost complete stoppage in the esophagus. At the age of eight she had typhoid fever, after which she had trouble with her eyes, and twelve years ago she became entirely blind. Her usual diet consists of vegetables and fruits.

Present trouble: The present trouble began forty hours ago while eating roast beef. Since that time she has been unable to swallow either liquid or solid food.

Examination: The patient was given 20 cc. of lipiodol to swallow, and the act of deglutition was observed in the fluoroscope. The lipiodol was seen to collect in the esophagus, about two inches (5 cm.) above the diaphragm, and no fluid entered

the stomach. The roentgenogram reading by Dr. Sam Brown was as follows:

"The esophagus presents an obstruction two inches above the cardiac end. The opaque medium presents a concave border, apparently due to an impacted foreign body, with a convex outline protruding upwards."

Operation: Without either local or general anesthesia, an esophagoscope was introduced, and the lipiodol was quickly removed, leaving the foreign body fully exposed. The latter consisted of an impacted plug of meat, which was removed piecemeal. The stricture was then observed and found to be an annular cicatricial stenosis with a diameter about that of a lead pencil. The patient made an uninterrupted recovery.

Comment: The picture was of value, inasmuch as the usual cone shaped outline seen in cases of obstruction was found reversed—i. e., with the convexity directed upward instead of downward, indicating the presence of a foreign body impacted above the stenosis.

At esophagoscopy the lipiodol was removed with greater ease than would have been the case with ingested barium, thus immediately bringing the foreign body into view.

Case 2.—D. C., aged 3½ years, son of a physician.

Previous history: The father states that the child has a habit of swallowing buttons. These have always passed by bowel without any difficulty.

Present trouble: On the afternoon of March 4, 1927, while playing with buttons the boy suddenly got up, made a face as if swallowing with difficulty and then cried for five minutes. There was no choking spell. At the evening meal, while attempting to eat mashed potatoes and tapioca pudding, the food was regurgitated after swallowing three mouthfuls. He was able to retain fluids but not solids. There was no change during the two following days.

X-ray: At his home in Dayton, Ohio, the patient was given three or four swallows of a half strength barium meal, and the roentgenogram taken one minute after the ingestion of the barium failed to show the presence of a foreign body in the esophagus, all of the barium appearing in the stomach and duodenum. The patient was not observed with a fluoroscope.

Lipiodol: The patient was brought to me two days after the onset of the symptoms. On admission to the hospital his temperature was 99.4°. He was given about 18 cc. lipiodol to swallow and was observed under the fluoroscope. Viewed laterally, the lipiodol was seen to divide into two streams in the upper esophagus, one stream flowing in front of an island-like obstruction, and the other behind the island. X-ray films showed some of the lipiodol opposite the third dorsal vertebra,[1] outlining a flat foreign body transversely placed. In the center of the foreign body a small circle, about one-quarter of an inch in diameter, was observed. Within this circle several small dots were seen, which were interpreted as thread holes in the button.

Operation: Without anesthesia, the foreign body was removed by the aid of an upper end esophagoscope, and found to be a large, thick, triangular shaped bone button. The patient's recovery was uneventful except that a petechial rash appeared on the day after the lipiodol was given. The rash was quite definite and was distributed on the forehead and cheeks. It disappeared in about a week.

A roentgenogram of the button after its removal showed that the small circle and holes seen in the original picture were rendered prominent by the lipiodol, which adhered in the crevices. After immersing the button in ether to dissolve the lipiodol, the sharply outlined circles disappeared from the roentgen film. (Fig. 1.)

Comment: In this case, lipiodol revealed the presence of a foreign body in the esophagus after the ingestion of a barium mixture had failed to demonstrate the presence of such a body. The adherent lipiodol partly outlined the foreign body and told something of its shape and nature. The lipiodol being tasteless was swallowed by the child without objecting. The petechial rash may have been due to the ingested iodin, but caused no particular discomfort.

Note.—In a recent case to obviate iodism, I gave a patient brominized oil, bromipin, instead of lipiodol, to show the presence of a foreign body, and found that the brominized oil, which is nontoxic, may be substituted for the iodized oil.[1] (Fig. 2.)

ESOPHAGOTRACHEAL FISTULA.

Case 3.—Henry S. (L 10187), colored, aged 45, was referred to me for examination by the medical service of the Cincinnati General Hospital, with the diagnosis of esophagotracheal fistula. This diagnosis had been established when the patient had swallowed a barium meal, some of which had entered the trachea and bronchi by way of the esophagus. However, the exact site of the fistula had not been demonstrated.

Examination: Left vocal cord paralyzed and in midline. Under local anesthesia applied to the trachea, the bronchoscope was introduced. Nine inches from the incisor teeth there was a nodular mass on the posterior wall of the trachea, partially obstructing the lumen. The mass bled on contact with the bronchoscope. A small nodule became detached and was obtained for study. The esophagus at the same level showed a nodular ulcerative mass on the right anterior wall. It also bled easily. A detached piece was taken for study.

Diagnosis: Carcinoma of the esophagus, eroding into the trachea. The fistula could not be seen either in the trachea or esophagus. A cotton tampon with a long string attached was inserted into the esophagus below the tumor mass, and the esophagoscope was withdrawn, leaving the string protruding from the mouth. The patient then swallowed lipiodol and in the fluoroscope it was seen to pass from the esophagus into the trachea and bronchi by way of a connecting fistula.

X-ray department, December 2, 1926. Reexamination of the previously described esophagotracheal fistula by the ingestion of lipiodol after the plugging of the esophagus below the site of the fistula, demonstrates a distinct connection between the esophagus and trachea with lipiodol in both bases of the lung field. Dr. Benzing. The tampon was then withdrawn by pulling on the string.

A gastroscopy was performed. The patient died about ten days later. At necropsy an esophagotracheal fistula was found.

Comment.—By plugging the esophagus below the fistula and having the patient swallow lipiodol, a distinct connection between the esophagus and trachea could be visualized to better

advantage than by the usual technic with barium. In a suspected case of esophagotracheal fistula it is preferable to employ lipiodol because the entrance of iodized oil into the bronchi causes no ill effects to the patient.

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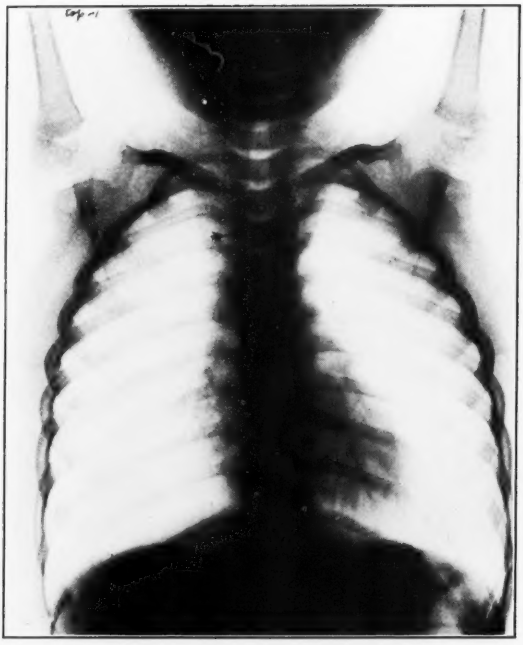

Fig. 1. Ingested Lipiodol adhering to holes and crevices in a button which had been swallowed by the patient. (Barium had failed to show the foreign body).

Fig. 2. Ingested bromipin arrested above and about a foreign body (sausage skin) impacted in a strictured esophagus in a boy nine years of age.

Open Safety Pin in the Stomach, Then Point Downward in the Esophagus: Removal.

By Samuel Iglauer, M. D., F. A. C. S.,

Cincinnati.

When an open safety pin is swallowed and remains in the esophagus, it is almost an invariable rule to find it lodged with the point upward. On mechanical grounds it is readily understood that for a safety pin to enter the esophagus, the spring coil must usually precede the point of the pin, which otherwise would tend to catch in the esophageal wall. The mechanism closely resembles that involved when a fish swallows the hook. On very rare occasions, however, a safety pin may be found in the esophagus in the reverse position—i. e., with the coil upward and the point downward.

Dr. Jackson in a personal communication has very kindly furnished me with an analysis of his records as follows: "We have complete records of one hundred and sixty-four safety pins removed from the air and food passages. The one hundred and sixty-four safety pins were taken from one hundred and fifty-eight patients, some of the cases having more than one pin, in some instances two, and in other instances three, and in one case, four pins. There were one hundred and fifty-one cases in which the point was directed upward. In thirteen cases the point was downward, including ten instances in which the safety pin was point downward in the esophagus."

I have encountered two cases of this kind.

Case 1, reported in 1922, occurred in the infant daughter of a physician.

A roentgenogram taken on the day of the accident showed this pin open end downward in the lower third of the esophagus. At esophagoscopy curds of milk were found in the lower esophagus. Upon withdrawing the tube the patient regurgitated the curds and pin (one inch long), which fell upon the floor. The parents were certain that the child had not vomited prior to this.

The second case occurred recently. History: George G. (L 10414), colored, aged 7 months, was admitted to Pediatric Service of the Cincinnati General Hospital, November 26,

1926, with a history of having swallowed an open safety pin on the morning of the same day. Fluoroscopy and the roentgen film showed an open safety pin freely movable in the stomach. (Fig. 1.) I advised daily X-ray examination to see if the pin would pass through the bowel. Two days later, fluoroscopy and the roentgenogram showed the pin point downward in the esophagus on a level with the fourth and fifth dorsal vertebræ. (Fig. 2.)

Operation: Without anesthesia, a short esophagoscope was introduced. The pin was not seen until the instrument was partially withdrawn, the foreign body having been concealed by a fold of mucous membrane and having been overridden by the esophagoscope. The spring coil was seized with a forceps and the pin was pulled into the tube and withdrawn in that position. The pin was of small size, measuring 1 inch in length (2.5 cen.). There was a febrile reaction (102.5°) on the following day, but the child made a complete recovery.

Note by Dr. Graeme Mitchell.—"There is no evidence of vomiting of food in this case, before the pin was removed. The mother, who was frequently with the child and the nurses, noted no evidences of gagging, although the child at certain times was not under observation."

(In case 1, also, the parents were certain that the child had not vomited after swallowing the pin.)

DISCUSSION.

Several theories have been advanced to explain the presence of an open safety pin, point downward in the esophagus. It is conceivable that a small safety pin might enter the esophagus in the usual manner and subsequently undergo spontaneous rotation through 180°. As I have suggested,[1] it is also conceivable that a very small pin (with a dull point) might enter the esophagus with the point downward. It is scarcely justifiable to assume that a pin could be swallowed closed and subsequently open in the esophagus.

Manges was probably the first to suggest that an open pin might enter the stomach, from which it might subsequently be expelled by regurgitation and lodged in the esophagus, point downward. In a recent personal communication and publication, he writes:[2]

"The open safety pin is almost as common as the coin, and they vary in size from quite small to very large safety pins. The open end is usually upward, but in several instances the open end was downward. In one of these, the pin probably opened after it had been swallowed—at any rate, the pin was bent and the point was partly missing, so that its closure was insecure. In another instance, the safety pin was regurgitated from the stomach into the esophagus, with the open end downward."

This pin had been in the stomach for seven weeks, when vomiting attacks carried it into the esophagus, from which it was removed by Jackson.[3]

Comment.—Case 2 (reported above) confirms the contention of Manges that a safety pin found point downward in the esophagus may have come into this position after having been regurgitated from the stomach.

This, I believe, is the second case on record in which an open safety pin was actually seen (X-ray) in the stomach and subsequently was observed point downward in the esophagus as described. In neither of my cases, both of which were closely observed, was there any history of actual vomiting. There may have been simple regurgitation without vomiting.

Under the title of cardioesophageal relaxation, Robins and Jenkelson[4] have recently described the occasional occurrence of reflux of the gastric contents into the esophagus. Presupposing such an abnormally relaxed orifice, a foreign body might, on rare occasions, be carried from the stomach into the esophagus.

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Fig. 1. Open safety pin in the stomach.

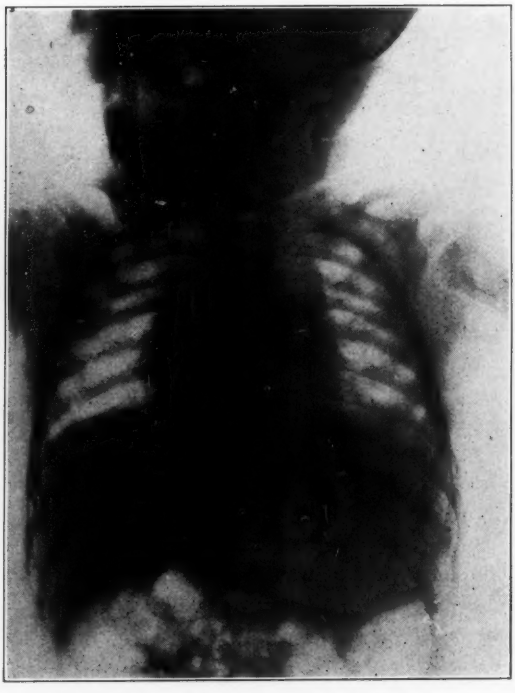

Fig. 2. Same, point downward, after it had been regurgitated into the esophagus.

Multiple Papillomata of the Esophagus: Report of a Case.

By Ellen J. Patterson, M. D., F. A. C. S.,
Pittsburgh.

A careful search of the literature reveals such a dearth of material on papillomata of the esophagus that the inference is that the disease is very rare or that the cases are undiagnosed and, therefore, unreported.

Delafield and Prudden,[1] in 1903, stated that small papillomata may occur singly or in considerable numbers throughout the entire length of the esophagus but that large ones are more rare.

Sir John Bland Sutton[2] states that papillomata may arise in any part of the alimentary canal and differ in character, according to the region in which they arise and the variety of epithelium that prevails. Papillomata are not uncommon on the tongue but rare on the mucous membrane of the esophagus.

Harlow Brooks,[3] in 1921, states that papillomata of the esophagus are infrequent and of little clinical importance. They are supposed to originate, when present, chiefly from aberrant tubular glands similar to those of the mucosa of the cardiac end of the stomach.

Veterinary journals contain reports of papillomata found postmortem in the esophagus of cattle.

In 1903, M. G. Petit[4] of Paris reported the case of a cow that died from inanition. The whole length of the esophageal mucosa was covered with papillomata so that nothing could get into the stomach but water, and that filtered through very slowly.

In 1905, R. J. Hicks,[5] London, reported a case of a two-year-old Shorthorn heifer that had attacks of choking over a period of two months. The attacks became more frequent, followed by marked distension of the esophagus after eating. At postmortem the entire length of the mucous surface of the esophagus was found covered with warty growths. Some of the growths were of the thickness of the finger and an inch and a half in length, not differing much in appearance from the papillomatous growths so frequently found growing on the skin of young cattle.

Bland-Sutton[6] states that the esophagus of the edible or green turtle is beset with processes which in structure resemble papillomas; some are pointed and 3 cm. long, with the points directed toward the stomach; others are merely low flat topped cones; each spine has an axis of loose connective vascular tissue covered with a layer of polygonal cells overlaid with a layer of epidermis almost as hard as horn. Edible turtles are strictly vegetable feeders and their chief food is the turtle grass.

There are two cases of papillomata of the esophagus reported in the literature.

In 1907, Chevalier Jackson[7] reported a case of single pedunculated papilloma of the esophagus in a male, aged thirty-six years. The growth was attached by a pedicle 3 cm. in length to the esophageal wall, 2 cm. below the interarytenoid space. Microscopic examination of the growth by Jonathan Wright showed it to be a papilloma.

In 1927, Kernan,[8] New York, reported a case of a child, aged ten years, with papillomata of the lower end of the esophagus, at 20 cm. from the teeth. Microscopic examination of a specimen from the growth showed papillomata.

A case of multiple papillomata of the lower end of the esophagus came under my observation in December, 1926.

V. T., a German, aged 66 years, was referred to me by Dr. Walter B. Harvey for difficulty in swallowing.

Upon admission to the Presbyterian Hospital, December 20, 1926, the patient gave a history of difficulty in swallowing solid food for eight weeks. He complained of great weakness, no pain, and regurgitated all solid food, though he swallowed liquids with difficulty.

Fluoroscopic examination of the esophagus with barium showed obstruction at the cardia, with some dilatation of the esophagus.

At esophagoscopy, the mucous membrane at the cardia appeared rough and fissured, with a slight degree of inflammation. (Fig. 1.) A specimen was removed with specimen forceps and sent to the laboratory for microscopic examination.

After esophagoscopy, the patient could swallow soft food easily and was discharged the following day, December 22,

1926, with instructions to report back to the hospital in one week.

Histologic examination was made by Dr. Harold G. Little, who reported as follows:

The epithelium is thickened slightly more than normal. The surface layer is flattened but shows no definite keratinization. The cells of the basal layer of the epithelium are elongated and apparently the layer is growing fairly rapidly. There is an occasional mitotic figure present in the basal layer. There is no rupture of the basal membrane or invasion of the underlying connective tissue. There is a slight amount of chronic inflammation in the connective tissue, as shown by the presence of lymphocytes and endothelial leucocytes. The connective tissue is normal in amount and the blood supply is

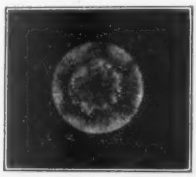

Fig. 1. Sketch of the endoscopic appearances of multiple papillomata of the esophagus as seen endoscopically in a man aged 66 years.

adequate. There is one small area of hemorrhage in the connective tissue. (Fig. 2.) Diagnosis: Papilloma.

The patient failed to report to the hospital in a week, as instructed, but returned January 26, 1927, looking very much emaciated. He had been able to swallow well for two weeks after the esophagoscopy, when swallowing became much more difficult and upon readmission he was suffering from "water hunger."

Gastrostomy was performed by Dr. Merle Hoon, to relieve the patient's immediate need of fluids and to afford us an opportunity to build up his nutrition. He put on weight rapidly and with the exception of an intercurrent attack of acute tonsillitis has made an uninterrupted recovery.

At esophagoscopy, without anesthesia, I have removed growths from the lower end of the esophagus four times, all

of which have been examined microscopically with the diagnosis of papillomata.

Clinically, this case obviously should be classed as one of multiple recurrent papillomata.

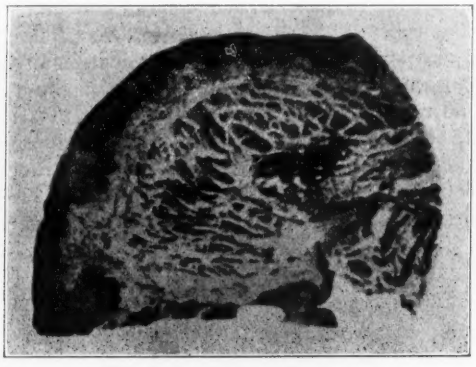

Fig. 2. Microscopic section of papilloma of esophagus.

CONCLUSIONS.

1. Papillomata of the esophagus, although rare, do occur, and if no one treated the esophagus without a diagnostic esophagoscopy, doubtless there would be fewer cases recorded.

2. Papillomata of the esophagus have a tendency to recur and should be removed as they recur.

3. Papillomata do not infiltrate, therefore, are best dealt with by superficial removal with cupped forceps, repeated as often as may be necessary.

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The Neglect of Direct Examination of the Esophagus in Cases of Dysphagia.

By Henry Hall Forbes, M. D.,

New York.

The short paper which I present is prompted by two recent cases of new growth involving the hypopharynx, which were seen in one of our large hospitals within a short time of one another and in which a diagnosis in one case was not made until within twenty-four hours of her death, and the second, who lived only a few days after the discovery of the diseased area. The first case was a female whom I was asked to see in consultation, at which time she was in extremis. At this time it was possible to see, by the use of the laryngeal mirror, a new growth involving the hypopharynx and the right pyriform sinus. This patient, I understood, had been examined by a laryngologist for dysphagia and had been told that no disease existed and that she could go home and forget it. Of course, we understand at that time the new growth had not been visible by use of the laryngeal mirror.

The second case comes under the care of my former chief of clinic, Dr. Frank R. Herriman, and through his courtesy I was enabled to examine the case. Here, as in the first case, no diagnosis was made. When brought to the hospital, the patient was so dyspneic that an immediate tracheotomy was necessary. The patient was a female, aet 41, and over weight. She had been treated for some time for indigestion, having dysphagia as one of her symptoms and later dyspnea from pressure on the trachea. The patient died of bronchopneumonia. The lesion found was a primary carcinoma of the upper end of the esophagus. How long the new growth had existed, we do not know, nor how long it would have remained undiscovered had not the acute dyspnea necessitated her removal to the hospital. At this stage the growth was inoperable.

My object in bringing this subject to the attention of the members of the American Bronchoscopic Society is to emphasize the fact that, notwithstanding our best efforts to assist the medical men as well as the surgeons, in the diagnosis and treatment, if you will, of all cases of dysphagia, apparent or

obscure, we have apparently failed to fully inform our own profession of the present completeness of our technic, of its freedom from danger as a diagnostic procedure and that our therapeutic efforts may be of some avail. I remember one case in which the daily press had been a greater factor than our efforts or the efforts of the medical press. Here a mother was told by her family physician to forget the fact that her child had swallowed a coin. She, however, at the end of a few days, when the coin was not passed, took her child to an X-ray laboratory. In the picture the coin was seen at the upper end of the esophagus.

I am quoting from a paper written by me in 1922: "A stomatologist seemed surprised when the suggestion was made that the use of the esophagoscope might help him in differentiating between cardiospasm and malignant disease. Special emphasis was made by me at the time that a death had occurred in a patient with malignant disease of the esophagus which had been treated as a cardiospasm for nearly a year."

Some years ago I felt that our work must be regarded as a scientific hobby, and, as one of our members aptly put it, "To be happy in this work, one must be an enthusiast." During the years that I taught bronchoscopy in connection with laryngology, my efforts were to interest as many as possible in the subject and even making the assertion, that no laryngologist should feel that his work had been fully covered until he could make a direct examination of the larynx and upper esophagus. In other words, I felt that his training must include a working knowledge of the use of the direct laryngoscope and esophageal speculum.

Our work is truly progressing. "Our work under local anesthesia and the development of the electrically illuminated instruments are now mere incidents." Our margin of safety is increasing. We all feel proud, I am sure, of the work of our fellow members and the original work of many.

This is all very well, and we know what should and what can be done. My own idea of the method to produce the best results, both for the patient and our own profession, is, first, continuance of our present progressive work. Due emphasis is still necessary for accurate histories, proper interpretation

of symptoms and preliminary roentgen-ray examinations in many if not all cases of dysphagia, and second, we recommend and urge the establishment, in every A hospital, of a department of bronchoscopy (naturally in the hands of the laryngologist), and that no examination of a patient suffering from dysphagia should be complete without a consultation and, if necessary, a direct examination by the bronchoscopist.

Endoscopic Treatment of Pulsion Diverticulum of the Esophagus.

By Charles J. Imperatori, M. D.,

New York.

Having accepted the postulate of Chevalier Jackson that "no endoscopic treatment is of any avail in the esophageal idverticulum so far as the removal of redundancy is concerned," and having seen the stormy postoperative reaction following the Otto C. Gaub operation, complicated by esophageal fistula, the operation proposed by Chevalier Jackson,* of inverting the sac endoscopically and encircling it with a ligature, appealed to the author, and in the course of the past year and a half three cases have been operated upon. The sacs were the following sizes:

Case No. 1—Depth 40 mm. by breadth 28 mm. S. F.

Case No. 2.—Depth 35 mm. by breadth 47 mm. T. H. C.

Case No. 3.—Depth 40 mm. by breadth 30 mm. H. P.

The first patient was 43 years of age, the second patient 74 years, and the last patient 57. In none of these patients following the operation was there more of a reaction than for twenty-four hours. Two of them were done under local anesthesia, one being done under ether anesthesia. A feeding tube was used in all the cases following the operation for from three to five days. Inspection of the ligated sac showed a reaction and edema in the first five days, which was followed by a pressure necrossi. The ligature in the three cases was a silk one and remained in situ for from five to seven days. The ligature in two patients remained on for six days and the other for five days. Gentle traction removed the ligature and in all three patients, most of the sac with it. Inspection of the site of the sac showed a superficially ulcerated area of about

*Peroral Endoscopic Laryngeal Surgery, page 550.

one-half to three-quarters of a cm. in diameter. At no time was there any evidence of esophagitis or any inflammatory reaction in the tissues of the neck.

The subdiverticular opening in all instances was dilated to the size of the large Jackson esophageal speculum, which is about 15 to 18 mm. in diameter.

Swallowing of alkalin fluids—that is, 1 per cent soda bicarbonate—is permitted and encouraged on the day following the operation—the tube still being in the esophagus.

Postoperative X-rays taken about one year afterwards, in two patients, showed no diverticulum or any further saculation of the esophageal wall. Functionally all of these patients are cured.

The method of operation is as follows:

Using a Jackson esophageal speculum or a modification of the Lynah esophageal speculum, the diverticulum is entered and contents aspirated, and then the sac is irrigated and aspirated with a saline solution, using a Yankauer lung abscess irrigator. Then mercurochrome, 2 per cent, is painted over the mucosa of the diverticulum. The beak of the endoscope is in the lowest part of the sac at the beginning of the inversion. With a Mosher grasping forceps, that has a locking device, an area of about 1 cm. of the most dependent portion of the sac is grasped and the whole sac is drawn into the endoscope. A slip noose is then thrown around the neck of the sac and tightened. Diathermy is now applied to that portion of the sac between the grasping forceps and a short distance above the ligature. Linear applications in the long axis of the sac should be the method of the application of the diathermy. The grasping forceps is now removed and the ligature is grasped by a forceps, extratubular, and withdrawn into the mouth. Holding the ligature, the esophageal lumen is now seen as in the examination of a normal esophagus. The endoscope is passed into the esophagus, thus dilating the subdiverticular esophagus.

A feeding tube is passed into the esophagus, through the endoscope, at least 20 cm. beyond the cricopharyngeus. The endoscope is removed and the usual procedure of placing the tube through the nose is done.

The after treatment consists in feeding per tubam for three to five days; the proper hygiene of the mouth and the swallowing of limited quantities of mildly alkalin cold water. Usually on the third, fourth or fifth day, depending on the reaction in the tissues of the sac, the feeding tube may be removed and the following mixture given in teaspoonful doses before and after feeding:

Bismuth subcarbonate, gr. 100; resorcin, gr. 20; glycerin, drams, 4; camphor water to make 4 ounces.

This mixture has been used for the past three years and is one similar to that used by Dr. Yankauer and suggested to the author by him when on service at Mount Sinai Hospital.

Case No. 1.—S. F., 43 years, Passaic, N. J., occupation, cantor. First seen August 3, 1925. An indefinite history of regurgitation of food, more marked within past eight months. There is no history of foreign body or any injury to the gullet. There was considerable deformity in the left neck, following each meal, and on palpation following the ingestion of fluids, fremitus and gurgling sounds.

On indirect laryngoscopy, Jackson's sign was positive in the left pyriform fossæ.

Diagnosis: Pulsion diverticulum.

These findings were confirmed by radiographs.

Operation as outlined above was done on March 4, 1926, at the New York Post Graduate Hospital, under local anesthesia.

Postoperative Notes.—March 5, 1926. Some slight soreness in throat. Able to swallow alkalin water.

March 6, 7, 8. Generally about the same. Temp., 99. Feeding per tube.

March 9. Removed feeding tube. Passed esophageal speculum into subdiverticular esophagus. Removed ligature. Small area of ulceration.

March 10 to 14. Eating some solids. Feels all right.

March 15. Examination of esophagus, using an esophageal speculum. No sign of pouching or diverticulum could be made out. Ulceration healed. Allowed to go home.

October 14, 1926, and April 13, 1927. Examination showed no evidence of any dilatation of the esophagus in the region of the cricoid. X-ray report shows a slight pitting at the region of the ligation. As to results, clinically this is a cure.

Case No. 2.—T. H. C., 74 years, Yazoo City, Miss., occupation, banker. First seen April 30, 1926.

About six years previously he had a foreign body, a chicken bone, removed from his upper esophagus with an expanding probang. Three years ago, he had an operation for appendicitis, and at that time it was necessary to pass a stomach tube but the attempt failed. During this period and for the past six years he has had on eating, a filling up sensation in the lower throat and always regurgitates food. On swallowing of fluids there is some slight deformity in the right neck, accompanied by fremitus and gurgling sounds.

On indirect laryngoscopy Jackson's sign was positive in the right pyriform fossæ. These findings were confirmed by radiographs. Direct esophagoscopy showed a saculation 15 cm. from the upper teeth.

Diagnosis: Pulsion diverticulum.

Operation as outlined above was done on March 4, 1926, at the New York Post Graduate Hospital, under local anesthesia.

Postoperative Notes.—May 5, 1926. Temp., 100. Feels very well. Sitting up in bed.

May 6, 1926. Temp., 99. Feeling very much better. Feeding through tube.

May 7, 1926. Out of bed.

May 11, 1926 Removed feeding tube and ligature. Esophagoscopy showed small ulcerated area at site of ligature. He is able to swallow very well. No gurgling or fremitus on palpation. Specular esophagoscopy very easily accomplished.

May 13, 1926. Permitted to leave the hospital.

May 14, 1926. Esophagoscopy—small ulcerated area at site of sac.

December 11, 1926. Esophagoscopy showed some slight redundancy of tissue that is protruding into the lumen of the esophagus in the region where the saculation was originally. No saculation whatsoever. Esophagoscopy very easily accomplished. As to results, clinically this is a cure.

Case No. 3.—H. P., 57 years, Union City, N. J., occupation, painter. First seen April 7, 1927.

Two years previously, while on shipboard, he swallowed a fish bone that was in situ several days, and dates his symp-

toms from this accident. On swallowing there is a slight deformity in the right neck and there is a fremitus accompanied by gurgling sounds.

On indirect laryngoscopy, Jackson's sign was positive in right pyriformfossæ. These findings were later confirmed by radiographs.

Esophagoscopy showed a saculation of the upper part of the esophagus, the bottom of the sac being 25 cm. from the upper teeth.

Diagnosis: Pulsion diverticulum.

Operation as outlined above was done on April 14, 1927, at the New York Post Graduate Hospital, under general anesthesia.

Postoperative Notes.—April 15, 1927. Very slight reaction. Temp., 99. Feeding through nasal tube and apparently very comfortable. Was able to swallow alkalin water without any discomfort.

April 17, 1927. Allowed out of bed.

April 18, 1927. Feeding tube inadvertently came out. Testing the patient for swallowing, he seemed to swallow normally and it was not replaced.

April 20, 1927. Allowed to go home.

April 22, 1927. Returned to office and ligature was removed with some part of the sac. As to results, clinically this is a cure.

CONCLUSIONS.

Two patients gave previous history of having had foreign bodies in the esophagus and dated their symptoms from that time.

As regards diagnosis, in all of the cases residual fluid, food or saliva was seen in the pyriform fossæ on the side of the diverticulum. This is Jackson's observation occurring in any obstructive condition of the esophagus, and particularly so when the obstruction occurs in the upper portion; the easy passage of the esophagoscope into the blind pouch and the difficulty to get into the esophagus proper; on palpation of the region of the suspected sac, after the ingestion of fluids, to feel a bubbly fremitus and to hear and feel a gurgle; some swelling after the swallowing of fluids in the middle part of the neck; the outline of the sac radiographically.

Results obtained by this method of obliteration of the sac are still subjudice. Apparently they are comparable with the other methods in vogue, but further reports of operations done, and especially so by other operators, are necessary to prove this method and its technic.

The length of time of operation being usually under one-half hour, the likelihood of its being done under a local anesthesia, the very slight local and general reaction, coupled with the ease that a practiced endoscopist can do the operation, recommends its trial and adoption over the more formidable procedure of the Gaub operation, insofar as shock, possible esophageal fistula and descending neck infection is concerned.

Of the three patients operated upon, one was done fourteen months ago and another ten months ago. They have remained symptomless since the operation and show no signs of return of the saculation. The third case is merely recorded, for he was operated upon but two months ago. This operation at this time appears to be as successful as the others.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of Monday Evening, February 7, 1927.

THE PRESIDENT, DR. ARTHUR M. CORWIN, IN THE CHAIR.

Primary Tuberculosis of the Sinuses.

DR. JOSEPH C. BECK: The patient was a negro, aged 17, who was admitted to the Research and Educational Hospitals of the University of Illinois in July, 1926. At that time he had evidence of interference with vision and came to the Eye Dispensary. However, after this one visit he did not return until seven months later, when he had lost a great deal of vision; the eyes protruded outward, he had pressure symptoms and gave a history of having had nasal hemorrhages. Sarcoma was suspected. The Wassermann reaction was negative, and there were no symptoms or signs of syphilis; examination of the chest was also negative. Radical surgical treatment was decided upon. Part of the middle turbinate body was removed for biopsy, and examination revealed tuberculosis, which had extended through the frontal bone and into the other structures. Shortly afterward he was radically operated upon by Drs. Lederer and Livingston by a supraorbital incision over both eyes. Broken down masses of granulation tissue and caseous material were found. The dura was thickened and covered with caseouslike granulation tissue but not interfered with. The wound was closed and drainage established intranasally and externally. The patient made an uneventful recovery, and there had been some improvement in vision, in ocular perception and in recognition of objects. The pus obtained from the frontal sinus region was sterile, but on a later growth staphylococci were found. No tubercle bacilli had been recovered, and animal inoculations had not yet been completed.

Examination of the eyes showed swelling of the nerves and later a choked disc, but there was no change in the eyeball. The characteristic facial changes were quite pronounced; due to the breaking down of the supraorbital bones and because of infiltration of the recti muscle, he was not able to hold the eye in proper position.

Skiagrams and photomicrographs were presented, showing the typical giant cells and tubercle formation.

Laryngectomized Patient.

DR. SAMUEL SALINGER: The man, aged 56, was first seen in November, 1926. At that time he was suffering with considerable dyspnea, due to a neoplasm which occupied the greater part of his larynx. Examination disclosed a rather uniform infiltration in the region of both ventricular bands, which seemed to extend down deeply into the larynx. A specimen was removed and biopsy showed an infected epidermoid carcinoma.

Laryngectomy was performed on December 10, 1926, under scopalamin, morphin and local anesthesia, without any general anesthetic. There was extensive infiltration of the larynx nad perilaryngeal tissues. On exposing the larynx, the thyroid isthmus was found attached to the larynx and infiltrated, the sternohyoid muscle on the right was involved, and several lymphatic glands seemed to be involved. The prognosis seemed so unfavorable that extensive operation seemed useless, but, nevertheless, all affected tissues were excised. There were adhesions due to a previous tracheotomy. The technic brought out by MacKenty was used. The patient was fed through an esophageal tube for four weeks. At that time the tube was removed, and it was found that there was a passage large enough to permit the swallowing of food. He had eaten without difficulty since that time and had gained twenty-five pounds in weight.

Histologic examination of the specimen removed showed a typical squamous cell epithelioma.

DISCUSSION.

DR. JOSEPH C. BECK thought the after care of such patients was the most important thing in connection with laryngectomy. The operation itself is not difficult, after one has mastered the points emphasized by MacKenty. Drainage after the operation to avoid infection is of great importance.

It was time that the X-ray was put in its proper place, and men should state that they had never seen a case of carcinoma remain cured after X-ray therapy. Laryngectomy gives such patients a perfect chance for permanent cure. If the patient

was promised that he would be able to speak following laryngectomy, that the operation would save his life and that he could be educated to talk by means of an artificial larynx, the operation would not seem so formidable.

Tracheal fistulæ often occur even after very careful operations, but Dr. Beck thought this was nothing against the operation. He asked about the condition of the glands of the neck, and said that cases had been reported in which the infection had disappeared following laryngectomy and removal of the glands. He believed the presence of such glands was not a contraindication for the operation.

Dr. Alfred Lewy said he had recently visited Dr. MacKenty in New York, and that he had worked out the after treatment in great detail. MacKenty had told him that he had never seen a patient recover from an extrinsic laryngeal carcinoma.

Dr. Salinger, closing, said Dr. MacKenty was very particular about the cases he selects for laryngectomy, but other surgeons had performed laryngectomies and removed infiltrated thyroids and glands, and gotten recoveries. Some cases have enlarged glands that are not truly carcinomatous. The glands should always be sectioned and examined carefully, for sometimes inflammatory conditions cause the same symptoms. He agreed that the after care was very important. The patient was a Slovenian who could not speak any English or German, and could not write or read any language but his own, so it was very difficult to give him speech instruction.

Foreign Bodies in the Throat.

Dr. Joseph C. Beck presented a patient who was referred to him because of complaints of pain in the throat. The history stated that an attempt had been made to remove some clips which were applied following tonsillectomy in another city. A skiagram showed six of these clips remaining in the throat. Dr. Beck had attempted to remove them under the fluoroscope but was unsuccessful. By direct illumination he was able to remove four of them, but two still remained in place, although the patient was unaware of this fact.

The case was reported to demonstrate that the presence of a foreign body in the throat would cause bitter complaint if

the patient knew it was there, but if unaware of its presence there would be no complaint. He believed the presence of the clips had nothing to do with the pain that was complained of.

DISCUSSION.

DR. LOUIS OSTROM, Rock Island, Ill., said that he had used these clips for many years, and that during the last fifteen years he had not had to visit a patient following tonsillectomy on more than six occasions. He had performed many hundreds of tonsillectomies and probably 400 radical quinsy operations. He has never had to use a ligature. After the tonsils are removed the hemostat is used. If that does not stop the hemorrhage within a reasonable time, he likes to use Stockton's needles for suturing the tissues. If the tissue is too solid to permit the use of this suture without causing bleeding, he applies the clips, making a circle around the blood vessel. In this way he has no difficulty in controlling the hemorrhage, and has never been called back to take care of hemorrhage in the next twenty-four hours. If the bleeding persists he sews in a pad. He uses the clips only when necessary, but knew of no patient who was carrying the clips who had any discomfort. He tried to take them out as soon as possible after operation, but explains to the patient that they can be removed at any time. He considered it perfectly safe to use them when necessary.

DR. W. E. GROVE, Milwaukee, Wis., said he had recently seen a patient in Milwaukee who had been operated upon by someone else several years before. A local tonsillectomy had been done. A needle had been left in the pharyngeal tissue, but the patient had experinced no trouble whatever.

DR. ROBERT GOOD thought it was dangerous to use such clips, for fear they might drop off and get into the larynx or be aspirated into the bronchi.

DR. BECK, closing, was glad to know that the foreign bodies did no harm if permitted to remain in the larynx.

The Relation of the Oral Articulative Movements of Speech, and of the Extrinsic Laryngeal Musculature in General, to Vocal Cord Function.

(ABSTRACT.)

DR. ELMER L. KENYON: The larynx, hyoid bone, lower jaw, tongue, soft palate and pharynx, together with their combined

intrinsic and extrinsic laryngeal musculature, constitute together, for all functions that involve the larynx, one united system of organs and muscles. Their functions are primarily those of animal life; secondarily, in man, they are employed also as organs of speech. The intrinsic and extrinsic musculature can act, whether for animal functions or for speech, only as one united muscular system. No other psychoneurologic means of action is provided. The peripheral physiology of speech is immediately based on the corresponding more basal physiology of the animal functions of the same organs. That the psychology of the peripheral speech function is also based largely on that of certain corresponding animal functions of these organs is probably also true. The extrinsic musculature is attached to the larynx almost exclusively by way of the thyroid cartilage, and thus may apply its laryngeal influence directly to the vocal cords, and, through cooperation of the thyroarytenoid muscles, presumably, to altering the details of vocal cord approximation. Studies of laryngeal function, apart from observation within the larynx itself, have been carried out on a wrong principle. Attempt to understand the action of so complex and intelligently cooperative a musculature by first studying the anatomy and then speculating as to how the muscles probably act, leads to inadequacy of result, as well as to error. It is possible to know the actual performance of such a musculature only by observation of what it actually accomplishes. Study by the author of the extrinsic musculature in actual action, by palpation of the upper anterior tip of the thyroid cartilage and adjacent structures, including the trachea, has yielded important physiologic information, both as to normal and abnormal speech. One important error in physiologic observation, that regarding the manner in which the vocal cords are stretched for purposes of elevating the pitch of the voice, has pervaded the textbooks of laryngology for at least 75 years. Actual observation, by palpation, of the action of the thyroid cartilage, shows clearly and definitely that elevation of the vocal pitch is produced not by tilting the thyroid cartilage downwards, as has been supposed, but by tilting it upwards. This is an act of the extrinsic musculature. By similar observation of the actual action of the extrinsic muscles, the whispered voice is found to be produced by joint action of

the entire intrinsic and extrinsic musculature, but with certain important differences from their action in the production of the loud voice. Cerebral excitation for the loud, as compared with that for the whispered voice, may possibly differ, not merely in degree, but also in kind. The interrelation between the movements of the mouth organs that determine the elemental sounds of speech and the approximation of the vocal cords for the production of voice are so immediately related as to constitute one complex physiologic act.

DISCUSSION.

Dr. Robert Good said that Professor M. Metfessel of the University of Iowa, Iowa City, is studying the singing voice. He takes moving pictures of the singing voice, and in that picture the slightest flatting or sharping of the voice is indicated. In addition to taking the moving picture of the voice, he takes a moving picture of the person who is singing and makes a record of the singing voice on a wire. In these three ways he analyzes the singing voice. The picture is taken by means of a small electric light and a small mirror. As the patient sings, the vibrations of the breath make the mirror vibrate, and the picture of the reflected light is shown on the screen. Dr. Good had visited his laboratory and seen the work. The wire is charged and runs from one spool to another, and will keep the record of the voice for hundreds of years. Dr. Metfessel had asked him what formed the vibrato and where it was. Dr. Good thought it was in the diaphragm and asked him to make moving pictures of the abdomen, but the Professor thought this was not the proper method of determining when the vibrato is made. He asked if Dr. Kenyon had any idea as to whether it was from the larynx, the diaphragm, or both.

Dr. Alfred Lewy stated that he had observed on his own larynx that as the pitch goes higher, not only the hyoid but the cricoid moves upward, and the interspace in front becomes smaller as the pitch rises. As he understood Dr. Kenyon, this was not in accordance with his observation.

Dr. Joseph C. Beck asked Dr. Kenyon to explain the whisper of the laryngectomized patient. Also, whether he had ob-

served or made use of the nasopharyngoscope and the Hays' pharyngoscope in watching the movement of the cords during whispering.

DR. ROBERT SONNENSCHEIN thought the Society was to be congratulated on having a man of Dr. Kenyon's type as a member. So far as he was aware, no one else had done such valuable physiologic work on the voice as Dr. Kenyon.

He asked whether the cricothyroid muscle acts as a tensor of the cords. It is unique in that it is supplied by the superior laryngeal nerve, all the others being supplied by the inferior or recurrent laryngeal nerve.

Referring to Dr. Good's report of the work of Professor Metfessel, Dr. Sonnenschein knew nothing about the method used by him, but there is nothing new about throwing the voice on the screen. It was done many years ago by Dayton C. Miller of Cleveland.

DR. KENYON, closing, stated that he was cognizant of the work of Professor Metfessel in Iowa City, but had no clear knowledge of what they were doing and did not wish to discuss it.

Replying to Dr. Lewy, he said that the physiologic facts were just beginning to be understood, and not much is definitely understood thus far, but in studying the action of the thyroid cartilage in the production of the voice one sometimes finds the thyroid cartilage moving up against the hyoid, and both moving up against the base of the tongue for the higher pitch effects. He thought there was nothing abnormal in Dr. Lewy's findings.

He did not know what the cricothyroid muscles do, but the extrinsic muscles are much more powerful and take hold of the cartilage in a much better way. It seemed to be a matter of cooperation of many muscles, and just what was the function of the cricothyroid he did not know. The old conception of the laryngologists was due to the fact that they did not conceive of them being extrinsic muscles at all, but that the cricothyroid was the one muscle that could stretch the vocal cord.

He knew very little about the speech of the laryngectomized individual, as he had never had an opportunity to study it in

a positive way. Dr. Grossman many years ago made tests and thought he found that somewhere in the lower part of the pharynx the tissues come together in such a manner that a vibration is possible. Then, by forcing the air out of the stomach, there was a sort of basal whispered sound.

He had given no thought to the production of the vibrato and did not know how to explain it.

Primary Chronic Hypertrophic Mastoiditis: Case Report.

ABSTRACT—INAUGURAL THESIS.

DR. HORACE R. LYONS: A male, aged 57, was examined on August 30, 1924.

In 1894 the patient had pulmonary tuberculosis. In 1912 he had scarlet fever, cholecystectomy and repair of ventral hernia, followed by a left antrum infection and empyema. In 1921 the patient had influenza, complicated by marked edema of both lungs. In 1922, following a very severe attack of influenza in 1921, the patient had a recurring, severe pain in his left ear. During the severe attacks of otalgia, examination of his left ear was entirely negative. There was no redness of the tympanic membrane, no bulging, no fever, no mastoid tenderness on prolonged deep pressure. The first attack of otalgia lasted about 48 hours and was severe enough to incapacitate him. After this attack disappeared, he had no further trouble for several months, when he again had a similar attack of acute, severe left otalgia. Examination by several otologists again revealed no evidence of middle ear infection. The tympanic membrane was normal. There was no bulging or redness, no fever and no mastoid tenderness on pressure.

The X-ray revealed a few cells in the left mastoid at the level of the antrum and above the knee of the sinus, which showed a slight cloudiness as compared to the other cells in the left and those in the right mastoid. There was no evidence of breaking down of the cell walls. The physicians consulted advised against interference, but the patient had the sensation of fluid in the middle ear and demanded a paracentesis. The left external canal was cleansed as thoroughly as possible and a paracentesis was done. With the aid of very slight suction, several drops of clear, thin, colorless fluid came through the

opening into the external canal. A culture and smear were taken and proved that the fluid was sterile. The patient was completely relieved of the otalgia. The same symptoms again presented themselves in the Summer of 1923. The consulted physicians advised against operative interference. A simple mastoidectomy was demanded by the patient and was performed on September 14, 1923.

The operative findings showed that there were several mastoid cells at the level of the mastoid antrum which contained a thin, colorless fluid, which proved to be sterile by several smears and cultures. Also, there was no evidence of breaking down of any cell walls.

The postoperative course was uneventful, and the patient had complete relief from his pain until February, 1924, at which time he again had a very severe pain in the left ear, accompanied by deafness, and pain upon deep pressure over the upper third of the left mastoid. He again had the sensation of dropping of fluid in his middle ear. This attack lasted about 24 hours. In the meantime, the infection of the left antrum continued, and the patient consulted a rhinologist in Boston, who advised and performed a radical external operation on July 7, 1924. As soon as the patient was able he returned to his home in Colorado. The antrum infection completely cleared up and he had no further difficulty until September, 1924, when he again had an attack of severe left otalgia, at which time he came under my care.

On examination, August 30, 1924, I found the tonsils cleanly removed, the nasopharynx negative. The nose showed the septum practically straight; no pus was found. There was no opening into the left antrum in the inferior meatus. There was a scar on the left side above the alveolar ridge, evidently the incision made for the radical external antrum operation. Transillumination showed that the light reflex from the left antrum was the same as that of the right.

Ears: The right tympanic membrane was retracted and slightly thickened; otherwise normal. The left tympanic membrane showed several linear scars, apparently the effect of the repeated paracenteses. The hearing tests with tuning forks showed no difference in the acuity in the two ears. Over the

left mastoid was found a scar giving evidence of a previous mastoid operation. Upon deep pressure, the patient complained of pain over a small area at the level of the mastoid antrum.

A skiagram, taken August 30th, revealed a shadow involving approximately four mastoid cells above and posterior to the knee of the sigmoid sinus, at the level of the mastoid antrum. The pain persisted for two or three days, and examination showed the same findings as reported above. The patient wanted his mastoid again operated upon, which seemed justifiable on account of the very definite history and on account of the X-ray evidence that there remained a few cells in his mastoid which had not been exenterated.

On September 2, 1924, the left mastoid was opened through an incision following the scar from the previous mastoidectomy. The periosteum was retracted posteriorly, and the mastoid was attacked at once directly over the area that the X-ray showed contained cells that had not been previously drained, the purpose being to open the cells in question before any other part of the mastoid was operated upon, so that if fluid was encountered it would not be complicated by oozing from a large wound. The cells that gave X-ray evidence of cloudiness were immediately encountered, above the sinus and posteriorly. The cell walls were not broken down, were lined with membrane and contained a very small amount of clear, colorless fluid, not enough to collect for study.

Following this the area previously operated upon was exenterated, for two reasons: First, to make sure there were no more mastoid cells; second, to find, if possible, a channel leading from the cells containing fluid to the mastoid antrum. No other mastoid cells were found, and there was no evidence of a channel leading to the middle ear. The dura was not exposed nor was the wall of the sigmoid sinus. The mastoid tip was not disturbed. The mastoid wound was closed with a drain through the lower part of the incision. A paracentesis was done.

The entire postoperative course was uneventful. The patient gained two pounds while in the hospital. Subsequently, there was never any drainage through the middle ear, and the mastoid wound was allowed to close; and the patient left town

on September 17, 1924. Several communications from him, the last one in December, 1925, state that he is perfectly well and has had no recurrence of his former trouble. It appears that this case was one of primary sterile secretory mastoiditis.

DR. G. HENRY MUNDT presented his entrance thesis entitled,

The Value of the Roentgenogram in Sinus and Mastoid Surgery.

BY G. HENRY MUNDT, M. D.,

CHICAGO.

The roentgenogram has found its way into otolaryngology because of its true worth. Few otolaryngologists today question its real value—a statement which could not be made a few years ago. The roentgenogram must be considered only one of the factors in a diagnosis and should not take the place of a careful examination of the patient. If the roentgenogram is to render maximum service to the otolaryngologist, it must be studied in conjunction with the other methods of examination.

To obtain maximum information from the films, it is essential that they be made with regular and constant methods. In this way only can one become competent in their interpretation. The otolaryngologist should develop the ability to interpret roentgenograms in order to secure full value from them. A written report from the radiologist may be of some assistance, but it is a poor excuse for a shadow box beside the patient during an examination. This is especially true in sinus work. MacEwen Smith says that many rhinologists and aurists have little confidence in roentgen ray findings because their films have not been interpreted with sufficiently detailed accuracy.

The two main factors in reading films are anatomy and pathology. Law says that the former is the more important, because the pathology is usually pretty well understood before the films are made. This has become especially true since we have gained some knowledge of the pneumatization of the temporal bone and the development of the sinuses.

In the interpretation of nasal films one must locate the sinus, note its size, apparent thickness of the walls and the presence

or absence of septa. In the maxillary sinus the relation of the teeth to the floor should be noted with great care. The relative position of the floor of the sinus to that of the nose will give very valuable information as to the handling of the case. If it were possible for the roentgenologist to give a report with accurate measurements and very detailed information, it might serve its purpose nearly as well as a few minutes' close study of the films by the surgeon. But no report can be as satisfactory as having the films in a shadow box beside the patient, so that they can be inspected while the nasal examination is being made.

Whether flat or stereoscopic plates are of most value is a mooted question, there being probably about an equal number holding each view. While the stereoscopic plates may be more spectacular, the writer questions very seriously whether the flat plate is not just as serviceable in the majority of cases. If it were possible to project mastoids and sinuses stereoscopically and secure the fine interpretation which one would expect ideally, I might favor stereoscopic films, but roentgen examination is not so perfected in detail as to show all the things one might expect.

Law says: "A film properly made gives definite information regarding the acuteness or chronicity of the conditions. Chronic purulent sinusitis shows a demonstrable osteitis while the hyperplastic type gives evidence of an edematous periosteum." Acute conditions, however, may show very little change. If the sinus is full of pus, it will probably be more dense than its fellow. In judging the density of a sinus or mastoid, the condition of its fellow must be the guide. It seems that in nearly all patients there is an equality in the mastoid and sinuses of the two sides, except in the frontal sinus, where there may be a great disparity in size and consequently in the thickness of the wall.

In chronic mastoiditis roentgen examination is of comparatively little clinical value in considering the advisability of an operation. It is possible to demonstrate a cholesteatoma and to determine whether eburnation or an eroding type of process is present, but this information has usually been obtained before the roentgenogram is made. MacEwen Smith says that

the position of the sigmoid sinus and emissary veins of the mastoid should always be noted. While this seems of comparatively little value, it may be of some operative assistance. In postoperative cases it is usually possible to demonstrate the remaining undrained cells and in this way to simplify an operation materially and improve the probability of a good result. This is one of the most valuable places for the roentgenogram in a chronic mastoid.

In acute mastoiditis it has been the custom to advise operation at any time that it is demonstrable that decalcification is taking place. Many methods have been devised to determine breaking down of the bone but none are so efficient in most cases as a series of films a few days apart. When it is noted that there is coalescence of the mastoid cells, it establishes without question that osteomyelitis is present and the mastoid may as well be opened then as later, as it will probably never clear up until it is attacked surgically. A film showing some cloudiness or blurring of the mastoid is of itself of no importance, as all acute mastoids will show some increase in density and many acute mastoids clear up. Let me reiterate that density in acute mastoiditis is not an indication for operation unless definite bone change is seen.

On the knowledge of pneumatization Law has arrived at a very interesting conclusion: "In acute mastoiditis, if large cells are found in the vicinity of the mastoid, operation can be no longer deferred than if these cells are very small. In the latter case, when mastoid symptoms are definite, it is safest to operate without delay." He further says, "The hemorrhagic type of mastoiditis is the most difficult and unsatisfactory for diagnosis; it occurs most often in large, fairly well developed mastoids. There is a peculiar transparency resembling polypoid condition, the septa are thinner than normal but are not lost; however, they may be absent deep in the region of the sinus plate. If mastoid symptoms are definite and the roentgen ray shows slight osseous destruction, be suspicious of hemorrhagic mastoiditis."

With a better understanding of Witmaack's theory regarding pneumatization, Albrecht has come to some very definite

conclusions. He feels that in pneumatization, variation from the normal shows lack of resistance of the mucosa and recommends the use of the roentgen ray not only because of its diagnostic value, but particularly for its prognostic value in acute suppuration. He reached this conclusion by a most interesting experience in a series of fifty-eight acute ear suppurations, which he describes as follows: "Fifty-eight acute suppurations; thirty-three healed without complications. Of these, twenty-six patients had normal mastoids, seven arrested pneumatization; twenty-three developed mastoiditis; of these twenty-one had normal mastoid development and healed readily. Two were arrested cases, of which one patient had to be reoperated for fistula. Two patients with partially arrested pneumatization died, one of meningitis and one of bulb thrombus eventuating in meningitis in spite of free operative exposure. Of the fifty-eight, there were in all eleven of partial or total arrest, and among these eleven were the two fatal cases and the one complicated case."

Kopetzky states that Heggre and Krauer, Wagers and Diehl have reported cases of acute mastoiditis of the coalescent type in which there was no evident involvement of the middle ear at any time. He considers the roentgenogram a most valuable aid in the diagnosis of this type of lesion. Certainly, if a primary mastoiditis is of the coalescent type, we have in the X-ray a most valuable diagnostic procedure.

In mastoid films the condition of the overlying tissue must be considered, as changes in the soft parts may cast a shadow on the film simulating a mastoid involvement, but if coalescence is watched for there will probably be no misinterpretation.

There are some roentgenologists who try to show labyrinth, middle ear changes and perforations. The value of such work is not generally accepted, and I am not competent to discuss it.

Sinus films are of great value, but here more than in the case of the mastoid close cooperation between the roentgenologist and the otolaryngologist is needed.

There should be no difficulty in securing good films of the frontal and maxillary sinuses, and ordinarily the anterior ethmoid cells can be very well shown. Because of the techni-

cal difficulties, many methods for showing the posterior ethmoids and sphenoid sinus have been devised, and I take it from the multiplicity of positions and methods that none is as yet a constant success; at least I have been unable to find constant satisfactory demonstration of these sinuses on X-ray films. Wherry advocates the oblique method of Rhese to show the sphenoid and ethmoid, and if he can get the information he seems to, it is indeed a well worth while procedure, but it has not been a success in the hands of many. In the opinion of some, Granger made a most valuable contribution in the method of showing the sphenoid and ethmoid, but his results are by no means easy of duplication and interpretation. His technic for sphenoid and mastoid has not been a success in the hands of a great many men.

Knox feels that the sphenoid sinus is best shown by the lateral position in practically the same plane as when the sella turcica is to be roentgenographed.

The Phaler method of making sphenoid roentgenograms with the film in the mouth, which was one of the first used, is still employed by some but has not been uniformly satisfactory. The Waters position is quite satisfactory for frontal and ethmoid, and the full face view for the maxillary.

It is better not to consider the interior of the nose in a roentgen report unless by chance there is a tumor or some other condition connected with a sinus. This statement is made to discourage the reports from roentgenologists that the septum is deflected or the turbinates enlarged, both of which conditions can be much better judged by anterior rhinoscopy.

A most interesting and apparently valuable contribution is that of Lean F. White on the measurement of the size of the optic canal by X-ray in optic neuritis. He concludes that the ability of the optic nerve to stand inflammation is to a considerable extent dependent on the size of the canal, and he recommends treatment based on the size of the canal.

Very slight sinus changes on the film may be of very great importance when considered with other symptoms. We are in the early stage of our knowledge concerning the development of the accessory nasal sinuses and I can see a great future in this line.

In conclusion, let me say that while in the future there is going to be great improvement in the visualization of sinuses and mastoids, the close study of films as they are made today is well worth the effort and time expended. I repeat, however, that the roentgenogram must be used in conjunction with the other elements of an examination in order to arrive at a diagnosis.

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DISCUSSION.

Dr. Joseph C. Beck said the essayist had overlooked a recently described means of examination of the sphenoid which was very reliable. With the plate placed beneath the lower jaw and the ray passed through the occiput, the sphenoid sinus will be demonstrated in a stereoscopic way. Dr. Reeves of North Carolina demonstrated such a plate, which showed not only the sinus but that the walls were necrotic and the sinus clouded. Dr. Beck considered this method a great contribution to the study of the sinuses. The Granger method had been made negative by many radiologists and rhinologists, who had proved it to be without value. He did not think the anatomy of the sphenoid was the same on both sides, and Prentice of Iowa City has shown a great variety of sphenoids going into the pterygoid fossa.

Dr. Beck agreed with Dr. Mundt that the pathology in many cases is not being determined, and thought this was due to the fact that the plates are not studied with magnification. If this

was done many valuable things could be made out in the skiagram.

DR. MUNDT, closing, hoped that Dr. Beck was right and that he could project the sphenoid in the way he mentioned in a group of cases. Dr. Mundt believed a thesis should follow the opinion of the writer, and he had tried many methods of projecting the sphenoid without finding any that was uniformly satisfactory.

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